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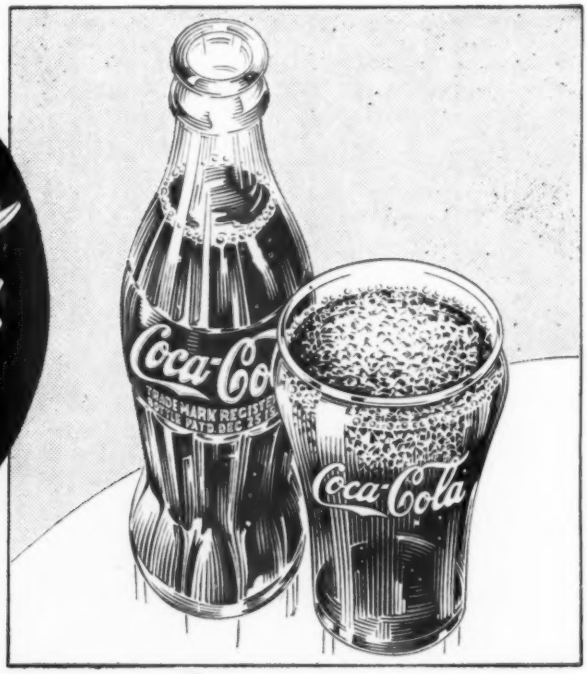

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VOLUME 217 SEPTEMBER 9, 1937 NUMBER 11

MASSACHUSETTS MEDICAL SOCIETY

Section of Tuberculosis

Presiding: Dr. Theodore L. Badger, Boston, secretary, and acting chairman.

THE CHAIRMAN: Dr. Pettingill, your chairman, has gone to Milwaukee and has asked me to open the meeting in his place. During the second part of the program I have a paper to read myself, and Dr. Morgan, from the Westfield State Sanatorium, will be kind enough to take charge at that time.

We are very glad to begin this program with a paper by Dr. Alton S. Pope on "The Role of Contact Examinations in the Control of Tuberculosis." Dr. Pope's paper will be read by Dr. David Zacks.

THE ROLE OF CONTACT EXAMINATIONS IN THE CONTROL OF TUBERCULOSIS

ALTON S. POPE, M.D.*

BOSTON

IN the face of a decline of approximately 75 per cent in the mortality rate from tuberculosis since the beginning of the century, it is disturbing to realize, as Drolet[1] has recently pointed out, that the fatality among recognized cases has not dropped appreciably during that period. This means in effect that the real progress in control has been effected not so much through improvements in therapeutic measures, such as collapse therapy, important as they are, as through a general decrease in the incidence of the disease itself and a corresponding reduction in the rate of infection. Such trends in morbidity and infection are, in fact, well established for several parts of the country. This is not the place to discuss the reasons why the apparent fatality rate is still approximately 50 per cent; as it happens, however, one factor which contributes to such a fatality is also a major factor in the perpetuation of the disease—late diagnosis.

We flatter ourselves that with facilities for x-ray and sputum examinations freely available in all parts of the State, we have provided everything necessary for early diagnosis; yet the proportion of minimal cases remains as low, and of far-advanced cases as high, as it was twenty-five years ago. At the Essex Sanatorium,[2] where all cases of pulmonary tuberculosis in the county have been admitted without selection as to clinical condition, the percentage of minimal cases during the past sixteen years has varied from 8.0 to 14.2, but has shown no consistent gain (Table 1). At the same time the proportion of far-advanced cases has shown a slight increase. There can be no question that with the means now at our disposal it is feasible to recognize pulmonary tuberculosis much earlier than was possible with the facilities generally available twenty to twenty-five years ago, yet it is evident that there has been no corresponding improvement in the discovery of early cases.

TABLE 1. *Cases of Pulmonary Tuberculosis at the Essex Sanatorium, 1921-1936.*

	Stage of Disease on Admission			
	Minimal %	*Moderately Advanced* %	*Far Advanced* %	Total Cases
1921-23	9.8	47.8	42.4	622
1924-26	11.1	35.6	53.3	794
1927-29	8.0	31.4	60.6	786
1930-32	14.2	28.2	57.5	772
1933-36	12.5	28.7	58.9	1043

The insidious onset of tuberculosis, with absence of symptoms and signs in the minimal stage, the reluctance of the average patient to seek medical advice until he is acutely sick,—and too often his refusal to accept a diagnosis of tuberculosis when it is first established,—together with a lack of "tuberculosis consciousness" on the part of the

Read at the annual meeting of the Massachusetts Medical Society, Section of Tuberculosis, Boston, June 1, 1937.

From the Massachusetts Department of Public Health, Division of Tuberculosis.

*Director, Division of Tuberculosis, Massachusetts Department of Public Health.

physician, all have been so well evaluated by Williams and Hill[3] and others that they need no further discussion here. Together they have served to nullify in large part the improvements in x-ray and laboratory procedures which make the recognition of early lesions possible. The present need is not for instruments or methods of greater precision, but for a practical technic for securing examination of that part of the population most likely to have tuberculosis.

In children the tuberculin test serves as an effective screen to separate those individuals already infected, and greatly reduces the cost of wholesale x-ray examination—the only practical way to recognize early pulmonary disease in that group. In Massachusetts that method has resulted not only in the discovery of a substantial amount of pulmonary tuberculosis in children of high school age, but over a period of ten years has raised the percentage of minimal cases in children admitted to the sanatorium from 6 to 50.[4]

The other method which has proved most productive in directing the search for unrecognized tuberculosis is x-ray examination of all family contacts of diagnosed cases of the disease. The tendency of tuberculosis to occur in families is one of its first recorded characteristics, and although this led to a hypothesis of heredity rather than contagion, the validity of the observation still holds. In most infectious diseases the highest incidence of secondary cases occurs among the family contacts, and this is especially true in a disease like tuberculosis, where infection depends upon continued, intimate contact. A number of estimates are now available on the incidence of pulmonary tuberculosis among the family contacts of patients with active disease, and the results have been surprisingly consistent. In New Haven among a group of 673 persons in contact with a case in the household, Edwards and Unzicker[5] found 59 new cases of phthisis, an incidence of 8.8 per cent.

In a recent field study in Williamson County, Tennessee, based on the examination of 453 persons in 150 families living in contact with known cases of tuberculosis, Stewart, Gass and Puffer[6] found that 8 per cent had definite x-ray evidence of pulmonary lesions.

The significance of such data from the standpoint of case finding is best appreciated in comparison with the incidence of tuberculosis in the general population. In the classical Framingham Demonstration in 1917 it was estimated that 9.5 persons per 1,000 had active pulmonary tuberculosis. Other estimates have placed the incidence at 1 to 10 cases per 1,000 population. In view of a decline of 67 per cent in the recorded tuberculosis mortality rate in Massachusetts since the Framingham study, it is obvious that there must also have been a substantial reduction of the incidence of the disease in the population at large. If the ratio of active cases to deaths has remained fairly constant, it is fair to assume a rate of some 3 active cases per 1,000 population in Massachusetts today. In comparison with this incidence, examination of 600 contacts of known cases in the outpatient departments of the Middlesex County and Westfield State sanatoriums revealed 46 new cases of pulmonary tuberculosis, a rate of 76 per 1,000, and of all forms of tuberculosis a rate of 210 per 1,000.

In the Chadwick clinics in Massachusetts, Zacks[7] reported a familial source of infection in 65 per cent of the school children found to have pulmonary tuberculosis. As a result of more recent studies he has found in the families of 320 such children evidence of 471 additional cases or deaths from tuberculosis during the past ten years, an incidence of 17 per cent.

Among some 27,000 high-school students tested in the school clinics from 1932 to 1934 the incidence of pulmonary tuberculosis was approximately 1 in 1,000, while in such children with home exposure to tuberculosis the rate was 25 per 1,000. If it were possible to obtain an accurate history of exposure in all cases, the search for tuberculosis in children might well be limited to the contact group, but unfortunately the history of such exposure is obtained only by diligent and tactful questioning, usually after the diagnosis has been established.

Second only in importance to its role in the discovery of tuberculosis is the influence of contact examination on the stage at which the disease is found. As previously pointed out, there has been practically no gain in the past fifteen to twenty years in the proportion of minimal cases reported; only by routine examination of family contacts has any substantial improvement been made. At the Middlesex County Sanatorium the family contacts of all cases are examined as part of the routine at admission; among the cases discovered by such examination some 58 per cent are in a minimal or moderately advanced stage, compared with 21 per cent among all patients admitted to the sanatorium.

Any method of treatment which halved the fatality of tuberculosis would rightly be hailed as epoch-making; yet that is a conservative estimate of the influence of early diagnosis upon recovery. Pneumothorax probably comes the nearest to reaching this standard, but its effectiveness is directly dependent upon the stage of the disease when found. How the stage at the time of diagnosis is reflected on the subsequent mortality is well illustrated in a study made of 400 cases admitted to the Rutland State Sanatorium between five and

six years previously.[8] Among the patients admitted in a minimal stage the subsequent fatality was 17 per cent, among those in a moderately advanced stage 41 per cent, and among those in a far-advanced stage 79 per cent. When it is pointed out that at least two thirds of all patients admitted to Massachusetts sanatoriums have far-advanced tuberculosis upon admission, no further explanation is needed for our failure to reduce the case fatality.

Yet even this is not the whole story. Pulmonary tuberculosis is spread only by patients who have tubercle bacilli in their sputum, and that means in large part by those with far-advanced disease. Not only have these people lost their best chance for recovery and a return to a normal life, but at the same time they have become the principal sources of infection for their families and friends. Without more exact knowledge of how long such patients have had open lesions before the diagnosis was made, it is not possible to get any quantitative measure of the part they play in the spread of tuberculosis, but in communities where the hospitalization of known active cases is the accepted practice, the individuals with unrecognized cases, who because of ignorance of their condition take no precautions, must be the major sources of infection.

With the rapid development of radiography and with the diagnostic services now available to physicians in the outpatient departments of county and State sanatoriums, and through the consultation clinics staffed by those institutions, x-ray examination is now available to any tuberculosis patient in Massachusetts, regardless of his financial status. A number of our sanatoriums are requesting the routine examination of all family contacts upon the admission of every patient. At such a time the problem of tuberculosis looms large to the family, and the matter of securing permission for examinations is simple. One county sanatorium has secured examination of approximately 90 per cent of all members of families of patients admitted. This procedure adds somewhat, of course, to the per capita cost of hospitalization, but at the same time it has brought more minimal cases to the sanatorium than have come from all other sources put together. Only when such examination of family contacts becomes the accepted practice of private physicians, as well as of all sanatoriums, can we effectively utilize our existing facilities for the control of tuberculosis.

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DISCUSSION

THE CHAIRMAN: The discussion will be opened by Dr. Roy Morgan.

DR. ROY MORGAN, Westfield: We have just listened to a very good paper by Dr. Pope. I think that there is nothing in it with which I disagree. There are, however, one or two points which I should like to stress. The first is the importance of examining contacts as soon as the original case is discovered. We have tried to carry on campaigns to examine contacts but have not had much luck with it. For some reason people will submit to examination a great deal more readily about the time a case is first discovered. At Westfield we have been finding about one out of ten contacts positive. When we encounter a positive case we always request the family physician to send in the other members of the family. If the original case is a new one, and the person has not been sick very long, we usually find the rest of the family all right; but, after a follow-up, cases begin to develop among them. The second point is the necessity of periodic examination, especially with adolescent persons. One patient was admitted about two years ago, all the members of whose family were negative at the time, but a year later we found one boy about seventeen with moderately advanced tuberculosis, and six months later another boy of the same age.

DR. GREENE S. FITZHUGH, Boston: How often should you follow them up? Every six months or year, or every three months?

DR. MORGAN: I think in adolescence I should follow up contacts every six months for two or three examinations, and then one a year, perhaps. It is a good deal a matter of judgment.

DR. IRA M. DIXSON, Stockbridge: It is always a pleasure to find one's convictions expressed by others. As a practitioner in a small community in the Berkshires it has become my conviction that tuberculosis has ceased to be especially a problem of the laboratory. We have become skilled in making the diagnosis, and it has come down to a question of control of the disease through the development of better contacts between families and general practitioners, of following through when the diagnosis has been made, and of getting the other cases that are probably incident and related to it. We now have a great bulwark in the practical handling of this matter—the branch of the Westfield State Sanatorium in Great Barrington. It is perhaps only due the Department of Public Health to know from someone in the field what a help that is. I think we have now three or four families in the village whose contacts are under supervision every six or twelve months, and it is a relief of my own responsibility which I greatly appreciate.

THE CHAIRMAN: I wonder if Dr. Zacks has anything further to add in relation to the comments which have been made.

DR. ZACKS: In discussing Dr. Pope's paper I want to stress a few points that come up when family contacts are examined. Of course the tuberculin test and the x-ray are the most important criteria. In mass examination you use the test as a screen and then x-ray the reactors. Occasionally you may miss a case in a child, or an adult for that matter, who has a negative test but has a lesion, and sometimes a far-advanced lesion. So that if the money is available, and particularly if the individual is known to be a contact, all the children and adults, even though they do not react in the test, should be x-rayed at least once.

The second question before this group is that of x-ray diagnosis. Naturally the diagnosis has to be made on the x-ray alone in most instances. No symptoms or signs are available. Now, there is a good deal of discussion as to whether a diagnosis should be made on x-ray evidence alone. I think the consensus of those who are in the work is that one can be thus made in a majority of instances. In infiltrative lesions the diagnosis should not be made immediately, unless it can be proved after subsequent x-ray; but in the typical case it is fairly easy to make it on the x-ray alone. When a diagnosis is made, the questions come up as to what to do about segregating, schooling, laying off work, and putting the patient under treatment. We have now had experience for about twelve years in following up these cases, and we know that, even with patients who are entirely asymptomatic when found, in most instances the diagnosis will be confirmed by the manifestation of symptoms if sufficient time is allowed. But by then it may be a little too late, because the disease may spread very rapidly. On the other hand, it may spread very slowly. So far as adolescents are concerned, we recommend treatment as soon as the lesion has been noted.

DR. MORGAN: The next paper on our program is "First-Infection Type of Tuberculosis in Adults" by Drs. Theodore L. Badger and Wesley W. Spink.

FIRST-INFECTION TYPE OF TUBERCULOSIS IN ADULTS

A Five-Year Study of Student Nurses at the Boston City Hospital

THEODORE L. BADGER, M.D.,* AND WESLEY W. SPINK, M.D.†

BOSTON

IT WAS only a few years ago, in 1928, that Heimbeck[1] called attention to the high percentage of young women in Oslo, Norway, entering the nursing profession who had never been exposed to tuberculosis. His studies[2] revealed that 81, or 21 per cent, of these nurses developed some form of tuberculosis during their course of training. This figure seems to us unduly high, and may be explained in part by the fact that he included 16 cases of erythema nodosum without other signs of tuberculosis. Heimbeck was the first to show that the first-infection type of pulmonary tuberculosis did not always take place as early in life as we had been led to believe it did in this country.[3] Among his nurses entering training, 52 per cent reacted negatively to tuberculin. All these reacted positively to tuberculin three years later at graduation.

Shortly after our study was started among the nurses at the Boston City Hospital, Geer[4] published a report showing that 30 per cent of the nurses at Anchor Hospital, St. Paul, Minnesota, were positive to tuberculin on admission to training and 98 per cent were positive when they graduated. Shipman and Davis[5] at the University of California, however, state that the majority of nurses tuberculin-negative on entrance remain so through training; but they had few tuberculous cases on their wards, and known contact with open disease was slight. In Minneapolis, Myers[7] in 1934 pointed out the high acquisition of positive reactors in nurses and medical students, and stressed the source of this as contact with open cases of pulmonary tuberculosis on the hospital wards. He stated that the ideal protection against tuberculosis was the absence of any exposure to the disease, and that such a condition was evidenced by a negative tuberculin test throughout life. Such an ideal situation seems difficult to attain at this stage of control of the disease, and a more practical point of view must be adopted.

Amberson and Riggins[6] in a recent five-year study of nurses in training at Bellevue Hospital in New York found that 62.9 per cent of 305 nurses were positive to 1 mg. of old tuberculin. Among 70 "negatives" followed through their three-year course of training, all but 10 became positive.

In January, 1932, the present study of tuberculosis in nurses at the Boston City Hospital was undertaken. Since then all nurses entering training have had complete physical examinations, quantitative tuberculin tests and stereoscopic x-ray examinations of the lungs. Only those failing to react to 1 mg. of old tuberculin have been declared negative, and yearly tests have been repeated on negative reactors. Stereoscopic x-ray examinations have been repeated yearly on all student nurses, and oftener when indicated.

TUBERCULIN TESTING

Our results have been very similar to the observations of others, insofar as tests on 470 nurses admitted to training in the past five years have shown

From the Thorndike Memorial Laboratory, Second and Fourth Medical Services (Harvard), Boston City Hospital, and the Department of Medicine, Harvard Medical School, Boston.

Read at the annual meeting of the Massachusetts Medical Society, Section of Tuberculosis, Boston, June 1, 1937.

*Junior visiting physician, Boston City Hospital.

†Former resident physician, Thorndike Memorial Laboratory.

271, or 57.7 per cent, positive and 199, or 42.3 per cent, negative to 1 mg. of old tuberculin. During the probation period some 34 per cent of the nurses failed in studies or resigned from school. Of 126 nurses who had finished their three-year training and who were 66 per cent positive to tuberculin on admission, 95 per cent were positive at graduation. In doing the quantitative test four dilutions were used which contained .001, .01, .1 and 1.0 mg. of old tuberculin. This technic was chosen in order to avoid large and painful reactions.

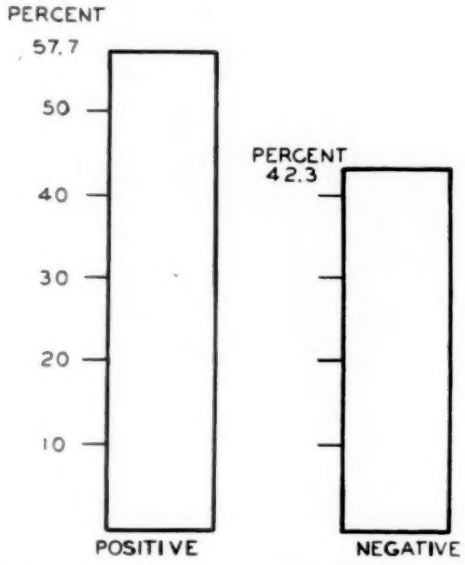

FIG. 1. Tuberculin tests on 470 nurses on admission to training.

TABLE 1. *The Distribution of 184 Positive Reactors to the Various Dilutions of Tuberculin*

Amounts	Reactors	
mg.	*no.*	*%*
.001	25	14
.01	63	34
.1	62	33
1.0	34	19

THE DEVELOPMENT OF LESIONS UNDER OBSERVATION

Let us now consider the 126 nurses who have completed their course of training, as well as 147 others who have remained in training one year or more. Among these 273 nurses 162, or 59.3 per cent, were positive to tuberculin on admission and 111, or 40.7 per cent, were negative. Eight tuberculous lesions have been observed in this entire group. One case developed among the 162 initially positive reactors. Seven cases developed among the 111 initially negative reactors. The one lesion developing among the positive reactors appeared after one year's training, and the nurse is still under sanatorium care, four years later. Of the 7 cases which appeared among the negative reactors, 3 nurses were allowed to remain in training, 2 did well on six months of sanatorium care, 1 has remained under treatment for nearly three years, and 1 is no longer alive. The protocols of these 8 cases are summarized in Table 2.

CASE HISTORIES

Case 1. M. H., a 19-year-old American girl, began her training in February, 1932, at which time physical and x-ray examinations were negative, but the tuberculin test was positive. She remained well for a year, when at the time of her routine x-ray check-up she developed an acute respiratory infection with a dry, nonproductive cough. Physical examination was negative, but x-ray of the lungs revealed a right apical lesion. Fever accompanied by pleurisy at the apices of both lungs appeared subsequently and she was transferred for sanatorium care. The disease has progressed for 4 years, and has been resistant to rest and collapse therapy.

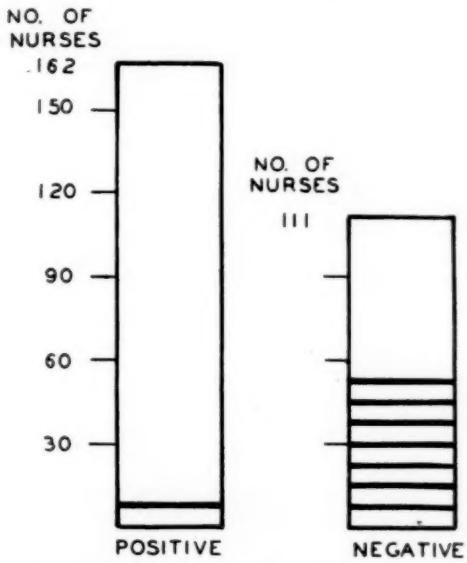

FIG. 2. Tuberculous lesions appearing in initially positive and negative tuberculin groups. (Each horizontal line represents one case.)

Case 2. J. F., an 18-year-old girl of French parentage, had a negative entrance examination in February, 1932. X-ray examination showed the lungs to be clear, and her tuberculin test was negative to 1 mg. Six months later the test became positive to 0.04 mg. of old tuberculin, but x-ray examination remained negative. A year later, in February, 1933, a routine x-ray revealed a greatly enlarged, right hilus lymph node and a minimal parenchymal lesion in the right mid-chest (Fig. 3). Physical examination at that time was entirely negative, and there were no symptoms suggestive of tuberculosis except for slight pleuritic pain in the right axilla, which lasted for 3 days. She was in other respects a normal healthy person. The x-ray film was interpreted as a primary tuberculosis of childhood type. She was persuaded with difficulty to stop training. The treatment prescribed was a clearly outlined regime of rest at home. For 4 months there was little or no change in the x-ray film, and she remained symptom-free. Then she contracted an upper respiratory infection and developed an acute pleurisy with effusion (Fig. 4),

TABLE 2. *Summary of Case Histories.*

Case	Diagnosis	Tuberculin Test on Admission	Symptoms	Sedimentation Rate	Physical Signs	Interval between First Positive Tuberculin and Finding of Lesion	Size of Lesion by X-Ray	Treatment	Tubercle Bacilli in Sputum	Result
				mm./min.						
1—M. H.	Pulmonary tuberculosis	+	Respiratory infection		0		3 cm.	Sanatorium—4 yr.	0	Under treatment
2—J. F.	Pulmonary tuberculosis Hilum nodes	0	Pleurisy—3 days	0.2	0	6 mo.	1 cm. and tuberculous lymph nodes	Home and sanatorium—10 mo.	0	Well
3—D. D.	Pulmonary tuberculosis	0	0	0.35	0	1 mo.	1 cm.	None	0	Well
4—G. McD.	Pulmonary tuberculosis	0	0	0.09	0	0	2 cm.	None	0	Well
5—M. C.	Pulmonary tuberculosis	0	0	0.6	0	0	1.5 cm.	None	0	Well
6—H. McC.	Pleurisy with effusion	0	Fever, pain and cough	0.8	Effusion	0	Pleurisy	Home and sanatorium—9 mo.	0	Well
7—H. C.	Pulmonary tuberculosis Arthritis	0	Fever—3 mo.	0.9	0	1 mo.	0.75 cm.	Sanatorium	0	Under treatment
8—M. R.	Pulmonary tuberculosis	0	Cough, fever—3 days		0	3½ mo.	8 cm.	Sanatorium	+	Dead

for which she had 5 months of sanatorium care. Six months later she married against advice, had a subsequent normal pregnancy, and has continued in good health. When she was last examined, 1½ years after discharge from the sanatorium, her parenchymal process had cleared, and the hilus node had disappeared. Examination has continued to reveal greatly limited expansion of her right chest with diminished breath sounds throughout that side.

Communication by letter November, 1936, stated that she was well.

Case 3. D. D., an 18-year-old Irish-American girl, started training in September, 1932, with a negative physical examination, negative x-ray of the lungs and negative

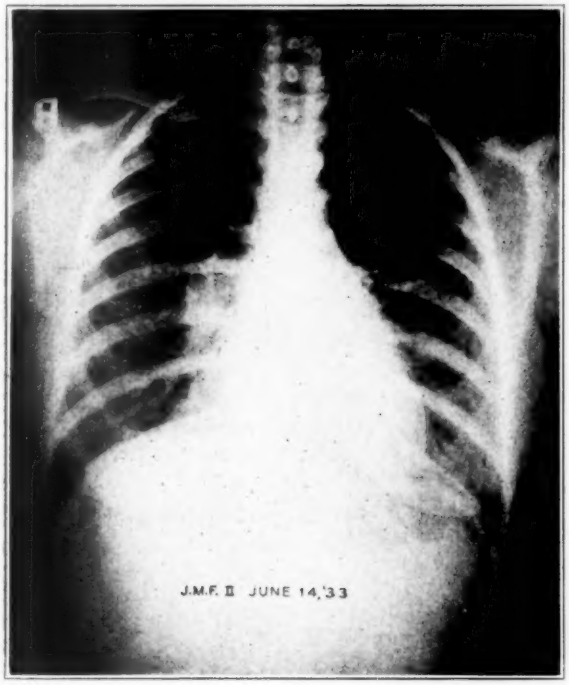

FIG. 3. Case 2. X-ray taken four months after the appearance of a small parenchymal lesion in the right mid-chest with greatly enlarged right tracheobronchial lymph nodes.

tuberculin test. Re-examination with tuberculin was again negative a year later but positive 2 years later, in July, 1934. A month after this first positive tuberculin test, her x-ray examination showed a lesion beneath the left first rib (Fig. 5), which was overlooked at the time. The diagnosis, therefore, was not made until April, 1935, from another x-ray film. Physical examination then was entirely negative, and because of her freedom from any symptoms and an excellent sense of well-being, she was allowed to remain in training, with monthly x-ray check-ups until the nature of the lesion was determined. She showed constant improvement in the x-ray film and is well today.

Case 4. G. McD., an 18-year-old American girl, was admitted to training in September, 1932. Her physical and x-ray examinations and tuberculin test were negative at that time. A year later the tuberculin test was still negative. In January, 1934, her routine x-ray showed a dense infiltration at the apex of the left lung, without physical signs or symptoms. Through an oversight her tuberculin test was not done until March, 1935, when it was positive. This girl was not removed from training because of the character of her x-ray lesion, but she was advised as to the findings and her intelligent co-operation was enlisted. She was kept under close survey and monthly

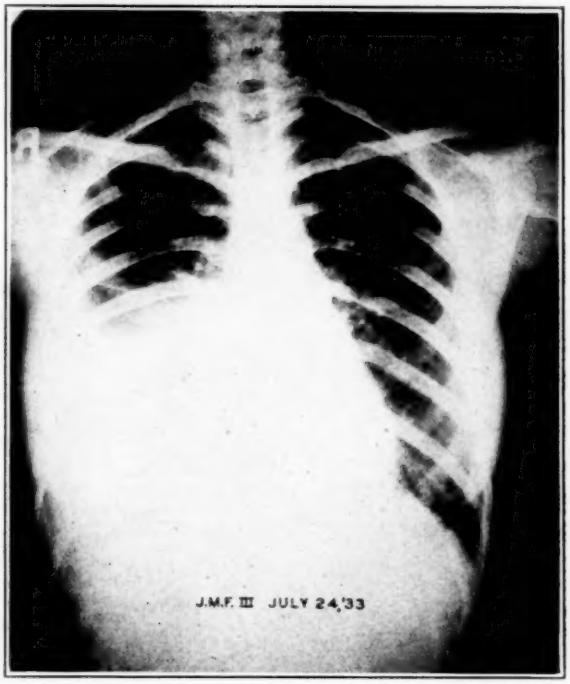

FIG. 4. Case 2. X-ray taken five weeks later, showing the development of pleurisy with effusion following an acute upper respiratory infection.

x-ray examination. No symptoms developed, and after a period of a few months the lesion began to diminish in size. In December, 1935, a year after its appearance, only

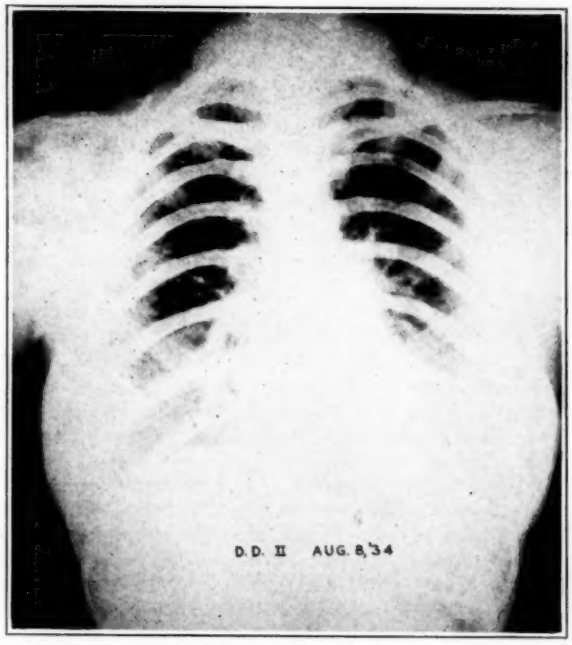

FIG. 5. Case 3. The lesion above the upper border of the first rib on the left was overlooked at this time and diminished somewhat over an eight-month period without treatment.

a dense fibrous strand remained at the apex of the left lung. She is well and working today.

Case 5. M. C., an 18-year-old American girl, began her first probation period in September, 1934, when she presented negative physical and x-ray examinations and a negative tuberculin test. Shortly after starting training she was forced to stop because of a hemolytic streptococcus throat infection. Re-entry to training 5 months later revealed a negative physical examination. At this time, however, she reacted positively to .001 mg. of old tuberculin and showed a minimal infiltration by x-ray in the periphery of the lung in the region of the right second interspace anteriorly (Fig. 6). Owing to her well-being

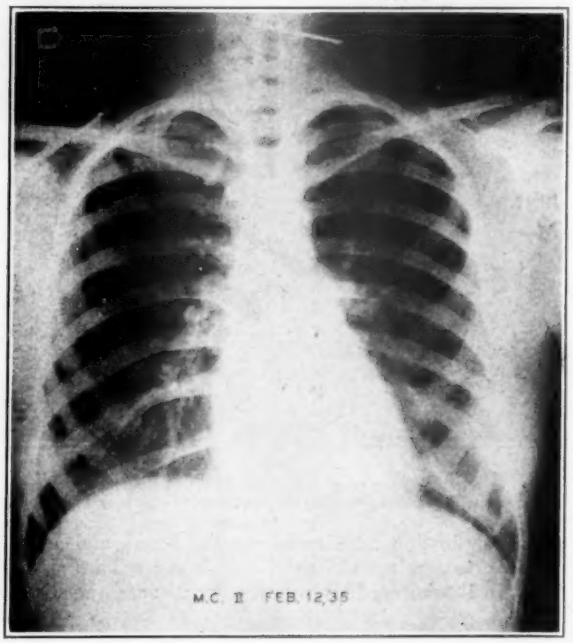

FIG. 6. Case 5. The plate shows a lesion in the periphery at the right first space anteriorly.

and the complete absence of symptoms she was allowed to remain in training with monthly x-ray check-ups. Her progress was an uneventful clearing of the process to a tiny area of residual scar tissue over a period of 8 months. She has remained entirely well to date (May, 1937), with the exception of a mild normocytic anemia in the summer of 1935.

Case 6. H. McC. an 18-year-old American girl, had a negative physical examination, negative x-ray examination of the lungs and negative tuberculin test when she started training in September, 1932. A year later, x-ray examination and tuberculin tests were still negative. A year and a half later, in May, 1934, she developed an acute pleurisy with effusion requiring a thoracentesis for relief of symptoms. The tuberculin test was positive at this time. After absorption of the fluid there could be detected by x-ray a very small parenchymal process in the periphery of the lung near the right second interspace, as well as mottling of the cap of the left lung. After 8 months of sanatorium and home treatment she was allowed to return to training with frequent check-ups and an uneventful course to the present.

Case 7. H. C., an 18-year-old Scotch girl, began her training in September, 1932, with negative physical examination, x-ray of the lungs and tuberculin test. In June, 1934, she developed fever, but did not report it until August 10, 1934. At this time the source of the fever was undetermined. She had lost 13 pounds in weight in 6 months and complained of fatigue and palpitation on exertion. After 2 months' observation in the hospital no cause for the persistent low-grade afternoon fever was discovered, but the tuberculin test became positive for the first time to 0.01 mg. In the third week of hospital care slight pain and swelling of both ankles and knees led to the diagnosis of rheumatic fever. Four electrocardiograms were within normal limits, but the sedimentation rate remained elevated. X-ray examination of the lungs was interpreted as negative. In October, 1934, she was sent home to rest in bed and did not reappear until February, 1935. Then, because of freedom from symptoms, a normal temperature and negative x-ray examination and electrocardiogram, she was allowed to return to training. Two weeks after resuming work she fainted. Because of a recurrence of pain in the right knee she was still considered to have rheumatic fever but failed to respond to therapy. Slowly her fever rose over a period of weeks. Physical examination of the lungs was repeatedly negative, but x-ray examination in May, 1935, showed an early pneumonic infiltration of the left upper lobe, and the sputum became positive for tubercle bacilli. Sanatorium care followed, and a successful pneumothorax

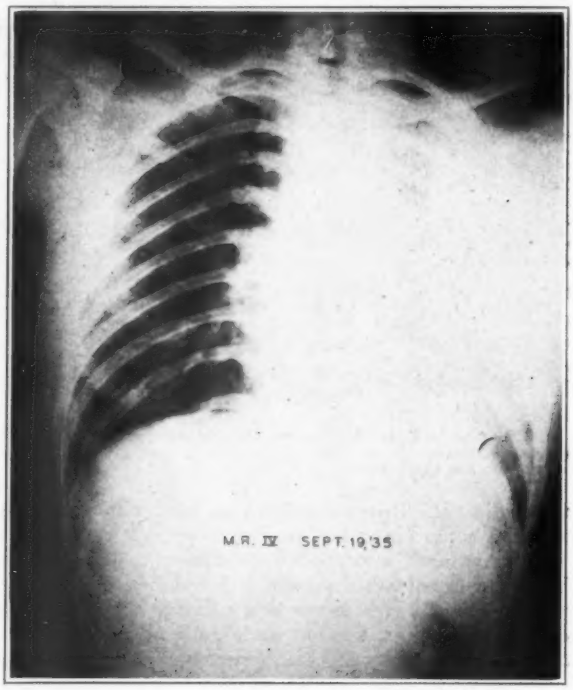

FIG. 7. Case 8. X-ray plate showing an acute pneumonic consolidation of tuberculous etiology developing in ten days under observation.

was obtained on the left. Tuberculosis of the sacroiliac joint developed, however, and an operation for fusion was done in January, 1936. Progress since has been marked by a persistent and variable fever without apparent satisfactory healing of her tuberculosis.

Case 8. M. R. an 18-year-old Irish girl, began training in September, 1934, with negative x-ray and physical examinations and a negative tuberculin test. There was no family history of tuberculosis. In May, 1935, she was admitted to the hospital for a transient, acute bronchitis.

Her tuberculin test was again negative, as well as x-ray of the lungs.

On September 8, 1935, she was admitted to the hospital with a chief complaint of burning micturition and frequency, stating that she was "perfectly well" until a week before admission, when she had a slight dry cough, fever, chilliness and nausea, all of which subsided. Examination of the lungs was negative. The urine showed only a few white blood cells. Her temperature was 101° F., pulse 100, and x-ray showed a faint fan-like infiltration extending from the hilum into the apex of the left lung. In another 3 days, signs of consolidation appeared high in the left axilla, the sputum was positive for tubercle bacilli, and the tuberculin test was positive to 0.01 mg. of old tuberculin. Eleven days after admission there was pneumonic consolidation of the upper half of the left lung without evidence of fluid (Fig. 7). She was then transferred for sanatorium care. Artificial pneumothorax was instituted in October, 1935; but there was a spread to the right side, and the process was never arrested. She died in December, 1936.

In considering these 8 nurses, we are particularly interested in the 7 cases which were negative to tuberculin at the start of training. These 7 nurses developed tuberculous lesions under observation, accompanied by the development of positive tuberculin tests. The diagnosis was made by x-ray in every instance except in Case 6, in which there was pleurisy with effusion. A more careful analysis of these 7 cases indicates that they may be grouped as follows: first, those with a process which was arrested with sanatorium care; secondly, those with a process which was arrested while they remained in training; and thirdly, those with a lesion which failed to respond satisfactorily to treatment.

Lesions Arrested with Sanatorium Care. Case 2 demonstrates a lesion characteristic of the childhood type of tuberculosis in an adult showing a small parenchymal patch in the periphery of the right mid-lung with enlarged right tracheobronchial lymph nodes. This lesion decreased when the patient was treated at home, but following an acute respiratory infection it lighted up and an acute pleurisy with effusion developed. The pleurisy then healed following five months of sanatorium care. She remains well four years later. In Case 6, the patient, who had a pleurisy with effusion, had an uncomplicated convalescence. She returned to work after nine months of sanatorium and home care, and remains well today.

Lesions Arrested while in Training. Cases 3, 4 and 5 were nurses whose lesions were discovered by x-ray examination. They showed no physical signs and presented no symptoms of their underlying disease process. They were all advised of the nature of their lesion as soon as it was discovered, and their co-operation was enlisted for close survey. No one of them was removed from duty for a single day because of the tuberculous lesion. All of them were under regular monthly x-ray observation until the character of their lesion was established. All showed variable but constant improvement in the x-ray film while remaining at work. All were advised regarding regular habits of living, high caloric meals, and extra rest if possible. No other treatment was given.

In Case 3, however, the lesion was overlooked in the x-ray film at the time of its first appearance and not recognized until eight months later on another x-ray examination. During the interval, when the nurse had no suspicion of the existence of the process *no* precautionary measures were taken. However, she remained free from any symptoms, and there was slight diminution in the anatomic extent of the lesion by x-ray during these eight months. Subsequently advised of the x-ray findings she secured more rest and nourishing food. More rapid clearing of the lesion appeared to follow, and she remained on duty without clinical or laboratory signs of activity.

Cases 4 and 5 present similarly benign lesions detected only by x-ray examination. Both were in nurses with no symptoms in any way suggestive of underlying disease. They remained in good health throughout training.

Any development of symptoms of activity in these nurses or extension of the process by x-ray would have resulted in bed treatment without delay. The character of these three lesions was particularly benign and now, from two to four years after their detection, there has been no clinical, laboratory or x-ray evidence of activation.

Lesions Failing to Respond Satisfactorily to Treatment. Case 7 was one with an unexplained fever of some eight months' duration, thought to be rheumatic fever because of joint pains. No satisfactory explanation of the fever was offered until the x-rays taken eight months after its onset revealed an acute pneumonic tuberculosis not detected by physical examination. Reviewing the x-ray plates, however, in the light of subsequent developments, we feel that in August, 1935, on admission to the hospital there was a small parenchymal lesion which, under prolonged bed-rest, did not heal. Hematogenous spread from this small lesion may have caused the acute pneumonic process and the sacroiliac disease that later developed.

Case 8, which appeared with so sudden and stormy an onset, was diagnosed and treated from the onset, but despite bed-rest and collapse therapy progressed unabated to a fatal termination in one year and three months after the onset.

DISCUSSION

These 7 cases of tuberculosis developing in young adult women are of interest because they represent the first-infection type of disease in adults previously shown to be free from infection by a negative tuberculin test. Such a test, however, does not always mean the absence of previous infection. In our total series of 470 individuals there are 6 with definite hilus lymph node calcification who failed to react to 1.0 mg. of old tuberculin. Calcification of hilus lymph nodes in young people usually means that tuberculous infection has been present. These 6 cases of hilus calcification must indicate old healed and obsolete infections. It is fair to assume, however, that except in such few cases as these, a negative tuberculin test in normal healthy people may be considered as indicative of the absence of tuberculous infection or tubercle formation.

Myers has shown that the first-infection type of tuberculosis is usually a mild and benign disease in children[9] and in young adults.[10] Our experience with this small group of nurses has led us to a very similar point of view regarding the nature of the lesion in adults. It should be emphasized, however, that a few of these small primary infections progress to destructive lesions despite an early diagnosis. The benign or destructive character of the lesion we believe can be determined only by clinical and laboratory studies, together with serial x-ray examinations taken at frequent intervals. It does not appear to us that either the character or location of most of these early lesions described is pathognomonic of primary or reinfection tuberculosis. Furthermore, the lesions observed in a single x-ray cannot be differentiated from bronchopneumonia, abscess or other early lesions of the lung. Only by serial periodic examination of x-ray films in conjunction with the clinical, laboratory and tuberculin tests can the tuberculous etiology of these lesions be established.

Having made the diagnosis, however, what seems most important is the early and lasting cure of these lesions detectable only by x-ray. Apparently some tuberculous lesions in young adults are so benign as to heal satisfactorily while the patient continues at his daily tasks. Treatment should consist of, first, the careful instruction of the patient regarding the underlying process, secondly, regular *monthly* x-ray and physical examinations until the character of the lesion is established, and thirdly, the intelligent adjustment of the daily program to include the maximum amount of regular rest and nourishing food. It must be remembered that the highest mortality age group in young women is eighteen to twenty-five years; therefore it is not possible to state at this time what the ultimate prognosis will be in regard to the lasting cure of these early first-infection lesions.

These 7 cases emphasize that, having made the diagnosis of tuberculosis, there remains a further responsibility for the physician. One must decide whether the existing lesion is benign or destructive and whether it is active or inactive. When such lesions are first detected, their nature is uncertain and can be revealed only by the closest study and observation.

We do not know whether a positive tuberculin test offers any degree of protection to persons exposed to open cases of pulmonary tuberculosis. Observations from this and other studies are of interest. Our figures (Fig. 2) show 1 case of pulmonary tuberculosis developing in 162 nurses reacting positively to tuberculin, and 7 cases developing in 111 initially negative reactors. Heimbeck[2] found 10 cases of tuberculosis developing among 186 initially positive reactors and 55 cases among 186 negative reactors. Amberson[6] showed 2 lesions appearing in more than 150 nurses tuberculin-positive at the start of training, and 6 lesions in 143 initially negative reactors. Myers[10] found that the majority of tuberculous lesions developed among those nurses where tuberculin tests were initially negative. Each of these investigators stresses the low degree of tuberculous morbidity in nurses positive to tuberculin at the start of training, as opposed to the high degree of morbidity in those who react negatively. In a group of young women such as these nurses the daily routine is the same for those initially negative or positive to tuberculin. Their exposure to open tuberculosis on the hospital wards must be approximately the same over a three-year period, yet we find more than five times as many cases of tuberculosis in those nurses negative on admission. In those nurses who have developed tuberculous lesions for the first time while under observation, it is too soon to know whether they have gained some degree of protection from their infection or whether they will develop destructive lesions in the future.

SUMMARY

1. A study is presented showing the development of tuberculous lesions in student nurses at the Boston City Hospital. On entrance to training 57.6 per cent of 470 nurses were positive to tuberculin. At the completion of their training 95 per cent were positive.

2. Eight cases of tuberculosis have developed during the past five years in 273 student nurses who remained in training a year or more.

3. Only 1 of these cases developed in the positive reactors on admission. Seven cases developed among the negative reactors on admission.

4. In the 7 cases that developed in nurses negative to tuberculin on admission, the lesions were of three types: first, those which became arrested with sanatorium care; secondly, those which were benign and became arrested while the nurses remained at work; thirdly, those which were progressive and failed to respond to treatment.

5. Regular, periodic x-ray examination is the most important procedure to be carried out in establishing the character of these early tuberculous lesions.

We are indebted to Dr. Paul F. Butler and Dr. Max Ritvo for their invaluable assistance in the interpretation of the x-ray films and to Miss Rose M. Foster, acting superintendent of nurses, for her co-operation in making this study possible.

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DISCUSSION

DR. MORGAN: The discussion will be opened by Dr. Julius G. Kelley.

DR. JULIUS G. KELLEY, Pocasset: The stimulation derived from listening to this excellent paper by Drs. Badger and Spink is multiple. First, one thinks of the large sociologic aspects, then come to mind many questions that are very urgently in need of answers. This paper and others definitely prove that the tuberculosis hazard is greater in the nursing and, I might add, the medical profession than in other groups of a similar economic level.

Stewart believes that there is a fundamental principle which governs the pathogenesis of tuberculosis in the body. He states: "Intrinsic immunologic factors of unknown chemical nature residing in tissue cells determine the type of tuberculosis which an infection with tubercle bacilli can produce; whereas, extrinsic environmental or extracellular factors (exogenous infection) or the accidental escape of tubercle bacilli from foci of disease (endogenous infection) determine whether the various pre-existing possibilities will materialize." In addition it is agreed that natural immunity of different peoples varies basically, while that of the individual may fluctuate under certain circumstances.

The problem of proving that acquired immunity through first infection is important enough to offset the dangerous potential of tuberculous allergy derived by the same process is a most vexing one. The paper just presented seems to indicate that primary disease confers an increase of immunity in this group of young females; but again a question: Do they, the previously allergic, go on through young adult life with less reinfection tuberculosis than those in whom allergy develops while under the stress and strain of the duties connected with a training school of nursing?

The first infection is definitely fraught with danger; about 3 per cent of these individuals, mainly children, develop the accidental and ofttimes fatal complications, such as meningitis, miliary disease, pleural effusion, and so forth, during the dissemination period of their initial process. We wonder if someone in the near future will have the fortitude to follow Heimbeck in the use of BCG among this group of young women with negative tuberculin reactions who are entering a hospital in preparation for nursing.

The other alternative or method of procedure is to prevent primary infection. This ideal seems impossible at the present time, but there is some evidence to prove that it can be accomplished in a much larger percentage of the population than is now evident. The first infection creates a definite susceptibility to phthisis, and pulmonary disease is the chief cause of mortality and the usual basis for increased morbidity. Therefore, I believe that we should exert every effort possible to prevent the spread of infection, for it is in this way that we can hope to eradicate this disease.

DR. MORGAN: Is there anyone else who cares to discuss this paper?

DR. ZACKS: I should like to ask for the name of the tuberculin that was used in these cases.

DR. BADGER: I very much appreciate Dr. Kelley's comments. I was interested concerning his reference to the work of Heimbeck. It is too soon to answer Dr. Kelley's problem. Not enough time has elapsed since the appearance of these lesions to know whether they will confer some lasting protection on the individual or eventually progress into destructive lesions. Another five years of observation is needed before conclusions can be definite.

The outcome of this study, with the one case of tuberculosis developing among the positive tuberculin reactors and the seven cases among the negative reactors, makes us lean a little toward the concepts of Calmette. He always believed that there was some degree of protection offered by the primary tuberculous infection.

An important point which I wish to stress is that in these lesions, whether they are first-infection or reinfection ones, a single observation by x-ray is not adequate. The lesions require a long period of observation before we can be sure about them. I think perhaps some people are given long periods of sanatorium care which this study demonstrates they can perfectly well do without. I offer this with a certain degree of caution, because I think that the general tendency is to allow many people to remain without the adequate treatment that they need. But there is obviously a group of patients with very benign lesions who get well, apparently, no matter what they do.

The tuberculin which we use has been the same throughout the entire study. It is Saranac Lake tuberculin, kept in concentrated form. Fresh dilutions were always prepared a few days before being used for testing.

THE CHAIRMAN: The final paper on the program today is one which is somewhat different from what might have been expected. It has to do with the results of surgery in tuberculosis from a medical man's point of view. It presents the end results of collapse of the lung in the treatment of tuberculosis. Dr. Julius L. Wilson, of the William Wirt Winchester Memorial Hospital, New Haven, Connecticut, is with us, and it is a pleasure to have him talk on "The Results of Collapse Therapy in Pulmonary Tuberculosis."

THE RESULTS OF COLLAPSE THERAPY IN PULMONARY TUBERCULOSIS

JULIUS LANE WILSON, M.D.*

WEST HAVEN, CONN.

COLLAPSE therapy for pulmonary tuberculosis has developed so rapidly in the past fifteen years, both in variety of procedures and in the number of cases treated, that there is a tendency both among the laity and among practicing physicians to expect successful application in almost every case. Without attempting either to advocate wider use of the newer methods of treating tuberculosis or to minimize the results obtained by them, I present the fruit of our experience at the Winchester Hospital in the past ten years and a brief review of other series of cases elsewhere. The question to be answered is: Armed with the pneumothorax apparatus and the ever-increasing resources of surgery, what can we offer the patient with pulmonary tuberculosis?

Artificial pneumothorax, perhaps better called "therapeutic pneumothorax," is the oldest, simplest and most effective means of collapsing the diseased lung. Fathered in 1888, by Forlanini of Italy who, thanks to antisepsis, was able to carry out the operation which Carson of Liverpool in 1819 could only dare to try on animals, independently advocated by John B. Murphy of Chicago in 1898, carried on by Brauer in Germany, and eventually controlled by Roentgen's x-rays and by Saugman's manometer, therapeutic pneumothorax was slow to make headway against the indifference and even opposition of those treating tuberculosis by time-honored methods. After the International Conference at Rome in 1912, where papers were presented on the subject by Forlanini, Brauer, and Dumarest of France, it became a part of the therapy of pulmonary tuberculosis; the long latent period was over. But opposition continued, and the strict indications which proposed particularly its limitation to strictly unilateral cases limited the usefulness of the method. However, as it was found that disease in the contralateral lung is not necessarily a contraindication, and as the technic was improved by the demonstration of the possibility of collapsing only the diseased portion of the lung, more and more of the patients were found suitable for this treatment, and had their lungs successfully collapsed. With the method of pneumonolysis invented by Jacobaeus in 1913 to sever adhesions and with the introduction of simultaneous bilateral pneumothorax, this application was further widened. Since the World War the opposition to pneumothorax therapy has ceased, and the method has been widely applied in every civilized country of the world. It has become not only a life-saving therapeutic measure for the individual patient, but also an important factor in the public health campaign to lessen infection in the community. Moreover, the practice of pneumothorax has taught us much about the physics and physiology of the respiratory system.

Surgical interruption of the phrenic nerve, first proposed by Stuertz as a simple phrenicotomy in 1911, and made practicable by Goetz and Felix in 1922 by the introduction of phrenicectomy or phrenic exeresis, has been widely advocated as a substitute for pneumothorax in cases where the latter is impossible and as a supplementary collapse procedure. The position of this operation as an independent method of treating pulmonary tuberculosis is still equivocal, with its enthusiastic advocates and at the same time its critics, who cannot grant that it is more than an auxiliary method. Its utility as a supplement to pneumothorax and other procedures is generally admitted to be valuable. Phrenicotomy, or crushing of the nerve, or alcohol injection to obtain a paralysis of the hemidiaphragm lasting for a few months has been gaining in popularity as the indications for phrenic interruption have become more exact, as the technic of the operation has improved and as greater knowledge of accessory branches has been acquired. This temporary paralysis as opposed to a permanent one is less apt to interfere with later procedures.

Extrapleural thoracoplasty, although as old as artificial pneumothorax, having been performed by Quincke for pulmonary tuberculosis in 1888, waited for many years upon the lessons learned by the clinician from the general adoption of pneumothorax therapy and upon the development of surgical technic and anesthetics. The work of Brauer and Freidrich established the parallel between pneumothorax and the resection of the ribs. Sauerbruch showed the importance of the paravertebral section of the ribs for the total collapse of one hemithorax. Thoracic surgery has developed as a special field since the World War. At present, progress in this field is rapid, with modification of the total thoracoplasty and the introduction of improved methods of partial or local-

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Read at the annual meeting of the Massachusetts Medical Society, Section of Tuberculosis, Boston, June 1, 1937.

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ized thoracoplasty. The future of the cases of chronic pulmonary tuberculosis surviving the initial attack and yet unsuitable for other methods of treatment is in the hands of the thoracic surgeon. The indications for such surgery are being made more exact, while at the same time improved technic permits operations on patients who only a year ago would have been considered hopeless cases.

The chief factor in the problem of treating pulmonary tuberculosis, as also in its prevention, is the tendency of the disease to break down the framework of the lung and to form cavities, which are then held open by the state of tension in which the lung exists throughout life. By what means the application of pulmonary collapse produces healing in such lesions is not settled. It was believed in the early days of artificial pneumothorax that the lung must be compressed to complete airlessness by pressure greater than atmospheric, but this procedure has been shown to be unnecessary in most cases. The diseased lung is in a state of both tension and trauma due to respiration, because it is in enforced uniform apposition to the chest wall. Measures of collapse therapy reduce both tension and trauma, and permit the retraction of scar tissue and the closure of cavities. None of these procedures heal in themselves, although with their help nature can and does heal more quickly and more effectively. It is important to remember that all methods of collapse or compression of the lung are merely valuable adjuvants to the old rest treatment. They do not replace and should not be considered as replacing rest and general treatment. All methods, to be effective, call for a longer or shorter period of thorough hospital or sanatorium care. Fortunately collapse therapy often shortens the period of bed-rest with great economic benefit to the individual and to the state, even though it does not shorten the whole period of treatment and medical care.

Before discussing the results in any series of cases treated by collapse therapy, one should set up a basis of the life expectancy in such cases without this treatment. Unfortunately this is very difficult, both because there is never any proper control for a given case of tuberculosis and because under modern sanatorium conditions such treatment cannot be withheld from any case merely as a control. However, we have some statistics based on past experience as to the expectancy of life of cases of tuberculosis with pulmonary cavities. Barnes[1] found that 90 per cent of such cases were dead within five years, with an average duration of life after the diagnosis of cavity of twenty-four months. These figures, of course, represent the results in a very unfavorable group in a state sanatorium, but even under the most favorable conditions of rest treatment not more than a third of the patients with persistent cavities can survive for any considerable time. Brown,[2] reporting Trudeau Sanatorium cases, found 76 per cent of the far-advanced cases dead within five years. We can all recall the general attitude toward far-advanced pulmonary tuberculosis prior to the introduction of the general use of collapse therapy in state and municipal tuberculosis sanatoriums. The *spes phthisica* of the ancients has changed from a delusion of the hopeless to a reality whereby not one in five manages to survive as an invalid but many can look forward to returning to their families and occupations.

The armamentarium of collapse therapy is employed at the Winchester Hospital in West Haven according to a definite program upon a patient population which is largely in the far-advanced stage of pulmonary tuberculosis. As we accept cases in any stage of the disease, on admission 80 per cent of our patients are classed as far advanced (National Tuberculosis Association classification), 12 per cent as moderately advanced and only 8 per cent as minimal. Each case is taken up at a staff conference, after admission and the necessary examinations, to determine what form of treatment offers him the best hope of recovery. If collapse therapy is possible and a cavity is present, as shown by a persistently positive sputum and by x-ray, no time is lost in waiting to observe the result of rest treatment alone, but artificial pneumothorax is attempted at once. Failing to obtain a satisfactory collapse, we turn without delay to phrenicectomy or thoracoplasty in suitable cases. Under this program 61 per cent of our patients have received one or more forms of collapse therapy, the other 39 per cent consisting of nontuberculous pulmonary conditions, favorable closed cases and hopelessly extensive terminal cases of pulmonary tuberculosis. This percentage of application of collapse therapy is by no means radically high, as is shown by the report of Leslie and Anderson[3] of the Michigan State Sanatorium, where collapse therapy was used in 79 per cent of 1,124 patients (9.3 per cent minimal, 32.5 per cent moderately advanced and 58.2 per cent far advanced).

Therapeutic pneumothorax is the most readily applicable and the most effective form of collapse therapy. The immediate results with satisfactory collapse of the lung in controlling fever, cough and hemoptysis are generally good, as shown in Table 1. This is based on 120 cases started between 1927 and 1932, classified according to the stage of disease. The real test of the method is the ultimate recovery of the patient with his return to useful life. Table 2 is an analysis of the follow-up on 88 cases started more than five years

ago and now re-expanded for from one to six years. It will be seen that of these, 58, or 68 per cent, are working after an average period of collapse of three years and three months, and 14, or 16 per cent, are dead after an average period of collapse of two years and five months, with 16 still living but unable to work. This table also shows that although in 34 per cent of the cases the lung re-expanded because of progressive adhesions before re-expansion was desired, these cases after an average collapse of only twenty-seven months have done as well as or better than the deliberately re-expanded cases in which the collapse averaged almost two years longer. Reactivation in the collapsed lung has occurred in only 3 cases in this series, and only once in the deliberately re-expanded lung, whereas new disease has appeared in the contralateral lung during or after re-expansion in 8 cases. No attempt has been made to classify separately the cases of bilateral pneumothorax, of which there were 6 in this series, 2 now working, 2 under treatment and 2 dead. There were also 18 cases of supplementary phrenicectomy, of which 12 are working and 4 dead. Of 7 patients who wilfully discontinued their treatment prematurely, 3 are dead and 3 working.

A pleural reaction with accumulation of fluid occurs at some time, usually in the first six months, in approximately half the cases of therapeutic pneumothorax. One in 8 of these becomes purulent. Table 3 shows the incidence of such effusions in the 88 re-expanded cases as exactly 50 per cent. While the appearance of fluid in the pleural space often results at the time in a better degree of pulmonary collapse, in this small series of cases the ultimate result has been better with the patients who never develop fluid, as 77 per cent are working as opposed to 55 per cent of those who had the effusion. Four of those with tuberculous empyema recovered without operation and are well, while 2 are dead.

In the eight years from December, 1927, to January, 1936, artificial pneumothorax was successfully performed on 432 patients of which number 128 were continued less than three months for various reasons. Of the remaining 304 cases an effective collapse with closure of cavities and a negative sputum were obtained in 179, or 60 per cent; in

TABLE 1. *Effectiveness of Artificial Pneumothorax in Cases Maintained Over Six Months, 1927-1932.*

Stage	Result of Treatment						Totals	
	Effective		*Partially Effective*		*Noneffective*			
	no.	*%*	*no.*	*%*	*no.*	*%*	*no.*	*%*
Moderately advanced	8	89	1	11	0	0	9	8
Far advanced, A	18	82	4	18	0	0	22	18
Far advanced, B	50	83	9	15	1	2	60	50
Far advanced, C	10	35	10	35	9	30	29	24
Totals	86	72	24	20	10	8	120	100

TABLE 2. *Re-Expanded Pneumothorax Cases Classified According to Method of Ending Treatment, Present Condition and Average Duration of Collapse.*

Reason for Re-Expansion	Present Condition									Totals		
	Working			*Under Treatment**			*Dead*					
	no.	*%*	*mo.*	*no.*	*%*	*mo.*	*no.*	*%*	*mo.*	*no.*	*%*	*mo.*
Physician's choice	33	65	48	11	22	40	7	14	29	51	58	42
Progressive adhesions	22	73	27	4	13	26	4	13	28	30	34	27
Patient's choice	3	43	—	1	14	—	3	43	—	7	8	—
Totals	58	66		16	18		14	16		88	100	
Av. duration			39			35			29			37

*Including 11 relapses: 1 in a deliberately re-expanded lung; 2 in prematurely adhering lung; 8 in contralateral lung.

TABLE 3. *Re-Expanded Pneumothorax Cases Classified According to Occurrence of Fluid.*

Course	Present Condition						Totals	
	Working		*Under Treatment*		*Dead*			
	no.	*%*	*no.*	*%*	*no.*	*%*	*no.*	*%*
No fluid	34	77	5	11	5	11	44	50
Fluid	24*	55	11	25	9†	20	44	50
Totals	58	66	16	18	14	16	88	100

*4 purulent.
†2 purulent.

87, or 28 per cent, the collapse was clinically partially effective; while in 38, or 12.5 per cent, it was ineffective. Complications were frequent but usually not serious. Fluid occurred in 172 cases (57 per cent), with 39 moderate and 15 large exudates of clear fluid, and 26 (9 per cent) of tuberculous empyema. A mixed tuberculous and pyogenic infection of the pleura occurred in only 7 cases, twice following pneumolysis and in the other cases resulting from bronchopleural fistulization. Extension of the disease to the contralateral lung occurred early in the treatment in 37 cases and late in 14, or a total of 17 per cent of the series. Spontaneous pneumothorax occurred in 18 cases. One patient died as a result of air embolism or so-called pleural shock.

Pneumothorax therapy was indicated and attempted in a total of 548 patients. However, in 116 instances (20.2 per cent) the pleural space was found to be obliterated by adhesions. In such cases, where definite indications for collapse therapy are present, phrenic interruption has been resorted to at once. The phrenic nerve has been evulsed on the corresponding side, resulting in a permanent paralysis of that leaf of the diaphragm. This operation was performed as the sole means of collapse therapy in 58 patients between 1927 and 1932. Table 4 shows the effectiveness of this operation in minimal, moderately advanced and far-advanced lesions, and its comparative failure in the more serious and extensive cases.

When this same series of cases is analyzed as to the results from five to nine years after operation, we find that 11 (19 per cent) are working and free of symptoms, 6 (10 per cent) are able to work with some symptoms, 11 (19 per cent) are living but unable to work and with positive sputum, 11 are untraced, and 19 (33 per cent) are dead. The degree to which the diaphragm rises after paralysis bears a direct relation to the effectiveness of this operation, as shown in Table 5. Thirty-seven per cent of those with a rise of 5 cm. or more recover, while with a rise of less than 5 cm. the number working decreases and the number dead increases. The location of the lesion in the lung, although at first considered most important, bears slight relation to the effectiveness of the operation. Cavities in the upper third of the lung were closed in almost as great a percentage of cases as those in the lower third, while lesions in the middle third were least benefited by phrenicectomy. Cases of temporary phrenicectomy or phrenic crushing were too few before 1932 for any conclusions.

Phrenic interruption is also used as a procedure supplementary to pneumothorax to diminish the vertical diameter of the pleural space when the lung is stretched between apical and basal adhe-

TABLE 4. *Effectiveness of Phrenicectomy, 1927-1932.*

Stage	Result of Treatment: *Effective*		*Partially Effective*		*Noneffective*		Totals	
	no.	%	no.	%	no.	%	no.	%
Moderately advanced	7	64	4	36	0	0	11	19
Far advanced, A	3	60	1	20	1	20	5	9
Far advanced, B	9	36	11	44	5	20	25	43
Far advanced, C	1	6	8	47	8	47	17	29
Totals	20	35	24	41	14	24	58	100

TABLE 5. *Cases With Phrenicectomy.*

Classified According to Rise of Paralyzed Diaphragm

Diaphragm Rise	*Well*		*Improved*		*Unimproved*		*Dead*		*Totals*	
	no.	%	no.	%	no.	%	no.	%	no.	%
5 cm. or more	7	37	5	26	2	11	5	26	19	35
Under 5 cm.	8	26	10	32	3	9	10	32	31	56
None	0	0	1	20	2	40	2	40	5	9
Totals	15	27	16	29	7	13	17	31	55	100

Classified According to Position of Cavity

Location of Cavity	*Well*		*Improved*		*Unimproved*		*Dead*		*Totals*	
	no.	%	no.	%	no.	%	no.	%	no.	%
Upper third	11	28	12	31	4	10	12	31	39	70
Middle third	2	20	3	30	2	20	3	30	10	18
Lower third	2	33	1	17	1	17	2	33	6	12
Totals	15	27	16	29	7	13	17	31	55	100

sions. It has been used with good effect in this way in 43 of our cases. In 8 a phrenicectomy has been done on the side opposite a therapeutic pneumothorax, and in 2 on the side opposite thoracoplasty operations. Phrenicectomy has been substituted for pneumothorax at the time of re-expansion of the lung in 20 cases.

Paravertebral thoracoplasty has been performed upon 36 cases of pulmonary tuberculosis, all far advanced, in 19 instances to collapse the lung and in 17 to replace a pneumothorax or empyema. Partial, apical thoracoplasties have been done in 8 other cases. Table 6 shows the results in the cases from three to nine years postoperatively; 47 per cent are free of symptoms and working, while 25 per cent are dead, with only 3 (9 per cent) of these dying in the first eight weeks. This unusually good experience with thoracoplasty is attributable to the careful selection of cases for operation, and even more to the advice and skill of Dr. Samuel Harvey and his surgical staff at the New Haven Hospital. The cases of partial thoracoplasty are too few and too recent for any conclusions, but our results with the operation are improving.

Combinations of the three principal procedures of collapse therapy are frequent and widely varied. To simplify the consideration of results I have purposely avoided subdivision of cases into those with pneumothorax plus phrenicectomy or with pneumothorax, phrenicectomy and thoracoplasty. The treatment is varied to fit the individual problem and to meet new conditions which may arise in each case.

There are numerous reports in the literature on the results of artificial pneumothorax treatment, as it is the oldest and most widely studied form of collapse therapy. However, it is difficult to compare these results because none take the same basis for classification; in the first place, some include all the patients upon whom the method was attempted, while others report only cases collapsed "successfully" or treated for various arbitrarily selected periods of time. Table 7 compiles some of the principal series reported in the past twelve years. It will be seen that in the inclusive series the percentage cured varies from 17 to 48, with a mortality of from 22 to 73 per cent, as compared with control series with from 5 to 16 per cent recovering and 60 to 79 per cent dying. In the series of cases of satisfactory collapse of the lung from 48 to 80 per cent recovered while 13 to 22 per cent died. Packard's series of re-expanded lung cases is interesting as showing 62 per cent recovered and working, with only 24 per cent relapsed or died and two thirds of the deaths occurring in the first three years after re-expansion. He also shows that

TABLE 6. *Results of Thoracoplasty Operations, 1927-1936.*

Operation	Living							Dead							Totals	
	I		Groups II		III		Total Living	First 8 wk.		9 wk.-12 mo.	2nd yr.	3rd yr.	4th-6th yr.	Total Dead		
	no.	%	no.	%	no.	%	no.	no.	%	no.	no.	no.	no.	no.	no.	%
Total thoracoplasty, replacing pneumothorax	8	47	3	18	2	12	13	1		0	1	2		4	17	47
Total thoracoplasty, original	9	47	2	11	3	16	14	2		2	0	0	1	5	19	53
Totals	17	47	5	14	5	14	27	3	8	2	1	2	1	9	36	100
Partial thoracoplasty	3	37	1	13	2	25	6	1	13	1	0	0	0	2	8	100

Group I. Able to work; bacillus-free.
Group II. Able to do some work; not bacillus-free.
Group III. Not able to work; bacilli in sputum.

lungs successfully collapsed two years or more seldom show recurrence.

Satisfactory reports of large series of phrenic nerve operations are very few. Table 8 gives a résumé of the results in two series of phrenicectomies without other procedures. It shows a favorable result with closure of the cavity and arrest of the disease in 27 per cent and 34 per cent respectively, while the mortality reported is 14 and 22 per cent. As a distinct aid to the rest treatment, these results cannot be ignored by the critics of the phrenic operation.

ing to death. The operative mortality itself is from 12 to 15 per cent in the first eight weeks in the series, but has been falling rapidly in the past few years owing to the universal tendency to divide the operation into multiple stages and to the improvement in anesthesia and surgical technic. Hedblom's own series of cases showed a higher recovery rate, 54 per cent; the Saranac Lake series, on the other hand, showed a lower recovery rate, 26 per cent. The alternatives faced by the patient are on the one hand the decreasing risk of the operation, offering a hope of recovery of working ability, and on the other the inconveniences of life, with a cough and a positive sputum, as a health-resort invalid, or what Dr. Lawrason Brown calls "a good chronic." For the poor man or the rugged individualist there seems to be no choice but to take the operation.

TABLE 7. *Artificial Pneumothorax. Results of Treatment by Various Authors.*

Name	*Patients*	*Well* %	*Improved* %	*Unimproved* %	*Dead* %
Matson, Matson and Bisaillon[4]	235 satisfactory collapse	48	44	—	22
	120 controls	5	9	—	66
Amberson and Peters[5]	122 after some years	26	—	—	48
	51 complete collapse	80	—	—	18
	52 controls	2	22		79
Naveau[6]	534	31	17	18	22
Peters, A.[7]	92 satisfactory collapse	45	—	16	31
Peters, Pope, et al[8]	400 effective collapse	67	—	—	6
	ineffective collapse	34	—	—	40
Packard[9]	100 re-expanded lungs	62	19		24
Hurrell[10]	100	17	4	6	73
Morgan and Saley[11]	107	60	15	10	6
Brown and Sampson[2]	515 all cases	31	25		43
	266 effective collapse	56	31		13
Brand, Edwards et al.[12]	3,021 cases	40	31		29
	2,750 controls	16	26		60

TABLE 8. *Phrenicectomy. Results of Treatment by Various Authors.*

Name	*Patients*	*Well* %	*Improved* %	*Unimproved* %	*Dead* %
Decker[13]	94	27	35	15	22
Nehil and Alexander[14]	272	34	35	17	14
	THORACOPLASTY				
Alexander[15]	1,154 collected cases	37	24		39
Hedblom[16]	168 personal cases	54	6	6	32
	3,762 collected cases	35	22	6	33

Large series of thoracoplasty cases have been collected by Alexander and more recently by Hedblom from reports all over the world. As shown in Table 8, the patient has a little better than one chance in three of recovering with ability to work, and about an equal chance of the disease progress-

DISCUSSION

A few general remarks may be made in respect to the results to be expected from collapse therapy. If one diagnoses a case of pulmonary tuberculosis and advises collapse therapy, too rosy a picture should not be presented to the patient. Optimism is justified in all except the most hopeless extensively bilateral conditions; yet these methods of treatment do not represent any easy short cut to health. In the first place hospital or sanatorium care with its treatment of the whole patient and education in how to live is essential during the period until the pulmonary lesion is under complete control, or at least until the sputum becomes negative. The general rest treatment cannot be neglected with patients being brought under pneumothorax treatment or having rib operations, and with phrenicectomy as the only form of collapse therapy, prolonged bed-rest is absolutely necessary. Pneumothorax must be maintained long enough to assure a permanent recovery, a minimum of two to five years after the sputum has become negative for tubercle bacilli. Phrenicectomy and thoracoplasty cases must be restored to active life gradually and under strict medical supervision.

There has been a tendency to apply the measures of collapse therapy earlier in the course of the disease. This is particularly true of pneumothorax and phrenic interruption. There is much to be said for this attitude, as minimal and moderately advanced cases are less apt to have pleural adhesions preventing collapse and have relatively few complications during the course of treatment. They can be restored to a normal life in a short time. It is, however, a fallacy to believe that a temporary phrenic interruption or from six to twelve months of pneumothorax will permanently arrest a lesion simply because it occupies only a small part of the total lung area. The necessity for prolonged treatment remains, even though practical control of the disease process can be obtained very promptly in favorable cases.

The feature of collapse therapy which appeals most to the practical-minded is the great economic saving effected. Hospital stays are shortened, saving the patient money and enabling the hospital to treat more patients with the same number of beds. This has been the reason for the wide application of therapeutic pneumothorax in France, and it has also helped the administrators of our state sanatoriums to reduce their waiting lists in a less expensive way than by new buildings. The patient also is benefited by being able to return to work earlier. He can work with a pneumothorax for several years, visiting his physician every two to four weeks for a treatment to maintain the collapse. This arrangement is obviously to the financial advantage of both patient and doctor. At the Winchester Hospital, with a very limited bed capacity, we have three outpatients for every inpatient on pneumothorax treatment, and the majority of these men and women are working full time. The chief advantage of thoracoplasty is that it may change a chronic invalid dependent on his family or the State to a self-supporting individual.

No figures showing the public health value of collapse therapy are available. However, since its goal is the closure of cavities and the ending of positive sputums, the widespread use of this treatment must be having a decided influence on the spread of infection in the community. This will in due time inevitably be reflected in a decrease of the morbidity and mortality of tuberculosis. More immediately, the prolongation of life by pneumothorax, phrenicectomy and thoracoplasty, whether the procedure is completely effective or not, has certainly been a factor in reducing the mortality from tuberculosis in communities where these measures have been applied.

SUMMARY

1. The program of collapse therapy in pulmonary tuberculosis has been applied to 61 per cent of the patients admitted to the Winchester Hospital.

2. Therapeutic pneumothorax has been attempted in 548 patients in a nine-year period, with some collapse of the lung in 432 and failure to locate the space in 116 (20 per cent). One hundred and twenty-eight cases were carried less than three months owing to various causes. A negative sputum was obtained in 60 per cent of the collapsed cases, and in only 13 per cent was the procedure completely ineffective clinically.

3. A study of the pneumothorax cases started prior to May, 1932, and now re-expanded shows that 66 per cent of the patients are now working and 16 per cent dead, the average period of collapse being three years. Whether the re-expansion was a result of progressive adhesions of the pleura or deliberate decision did not affect the result materially. The occurrence of pleural exudate did tend to make the end result less satisfactory. Recurrence of activity in the re-expanded lung occurred in only 3 cases, while the appearance of new disease on the opposite side occurred in 8 cases.

4. Phrenic nerve interruption alone with diaphragmatic paralysis was effective in 35 per cent of 58 cases, succeeding in direct ratio to the rise of the diaphragm and in inverse ratio to the extent and severity of the disease, with location of the cavity apparently of no importance.

5. Paravertebral thoracoplasty was performed upon 36 patients and partial thoracoplasties upon 8 others. Total thoracoplasty resulted in 47 per cent recoveries in from three to nine years, and

a 25 per cent total mortality, with 8 per cent immediate postoperative mortality.

6. The results of pneumothorax, phrenicectomy and thoracoplasty reported by various authors are summarized.

CONCLUSIONS

We can offer our patients with pulmonary tuberculosis a vastly improved outlook for recovery with the aid of collapse therapy. If pneumothorax can be successfully induced their chances of recovery are tripled. With phrenicectomy alone their chances are doubled if the disease is not too extensive. For the chronic tuberculous invalid, thoracoplasty opens the door to a useful life with the odds that he will recover better than that he will either die or not be improved. With patients with sufficient good lung remaining, and with the unknown quantity in the tissues which it takes to heal tuberculosis, combinations of these procedures can work what seem like miracles.

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DISCUSSION

THE CHAIRMAN: We are all indebted to Dr. Wilson for presenting a very interesting and comprehensive survey of the effects and the evaluation of collapse therapy in tuberculosis, and we are very glad to have Dr. Andrew Peters open the discussion.

DR. ANDREW PETERS, Springfield: The whole subject of collapse therapy in pulmonary tuberculosis covers such a wide field that it is very difficult to do it justice in the short space of time available. The very greatly increased application of this method of therapy in recent years—especially since 1930—makes an evalution of its results an extremely important matter, and we have not nearly such complete data as we ought to have and as many apparently think we have. Groups comprising large numbers of cases have indeed been reported, particularly from several institutions in Middle Western cities, but unfortunately most of them represent very recently discharged patients, and a considerable proportion (as in the recent report by Leslie and Anderson from Michigan State Sanatorium) are still resident institutional patients. To evaluate results by the classification on discharge is open to many serious objections, and when these results are compared with those of a previous period we have to remember that the official standards of classification on discharge have changed; for example, prior to about 1930 it was not permissible to give any patient with a pneumothorax a classification better than "quiescent." Furthermore, to evaluate any procedure on the basis of the number of patients subjected to it involves many logical fallacies. We have only to look back at the erstwhile popularity of tuberculin and later of heliotherapy for examples of such fallacious reasoning; yet as shown by some reported series of cases and even some official committee reports, high percentages of cases subjected to this or that form of surgical or collapse therapy are cited as though they furnished incontrovertible evidence of its advantages, and the obvious non sequitur is not perceived by the authors. Far too often do we hear it casually stated that so many data have been accumulated and recorded on so many thousands of cases in innumerable series reported, particularly in the case of artificial pneumothorax, that any further probing of results is superfluous and unnecessary. On the other hand, F. J. Bentley, in a recent survey of the subject, reviewing all the literature available together with the experiences of the London County Council, states in a Report of the British Medical Research Council (July, 1936): "It is doubtful if anyone at the present time could produce reliable data on 3,000 pneumothorax cases remaining well over a seven-year period." In spite of this, it seems difficult to interest clinicians in controlled statistical studies. One difficulty common to nearly all series, including that analyzed from a group of American institutions by the Committee on Artificial Pneumothorax of the American Sanatorium Association, is the necessity of lumping together cases treated or observed over widely varying periods of time, for example, two to twelve years, one to six years, and so forth. Unless one knows how many cases fall in each time period or interval the resultant composite figures are vitiated by important unknown variable factors. For this reason it seems desirable to some of us that new investigations be undertaken in which the cases would be properly grouped chronologically and followed over comparable periods of time, preferably not less than five years.

There are some outstanding very practical questions concerning collapse therapy in pulmonary tuberculosis, the answers to which are not by any means completely satisfactory and regarding which there is far from unanimity of opinion among sanatorium men and phthisiologists.

What proportion of general sanatorium admissions is suitable for pneumothorax therapy, for thoracoplasty, and for other collapse procedures? Under the last we should consider *phrenic nerve interruption.* Only a few years ago this measure was very extensively used, partly, no doubt, owing to its apparent simplicity and brevity, and the expectation in some quarters seemed to be that it would partially supplant pneumothorax, doing away with the necessity for frequent observation and long-continued refills, and would also come to be a common procedure in early cases, yielding a sufficient degree of pulmonary restriction and partial collapse to promote healing. Neither of these expectations has been realized, and this procedure now seems to be used less and less commonly by experienced phthisiologists and thoracic surgeons; where it is

used, the permanent operation of extirpation or exeresis has been almost entirely superseded by the temporary one, that is, crushing of the nerve. Reasons for the declining use of phrenic nerve operations may be several. As an independent procedure it has proved relatively ineffective in the treatment of extensive or severe disease and in most cases with early limited or less serious lesions its disadvantages outweigh its advantages. Exceptions may be noted in cases with fairly recent cavitation in the lower half of the lung where there are not extensive basal pleural adhesions and thickening. As a supplementary procedure to pneumothorax or thoracoplasty it has proved of very limited value, chiefly in that a relatively small proportion of pneumothorax patients have a considerable involvement of the lower part of the lung, the lesion either not collapsing satisfactorily under pneumothorax or tending to re-expand too readily because of diaphragmatic movement, or have a cavitary lesion definitely held open by tension on an adhesion or band of adhesions attached to or near the diaphragm.

In a paper read before this section in Boston five years ago, I ventured the opinion that the proportion of general sanatorium admissions that might be considered "good prospects for pneumothorax therapy" would run about 15 to 25 per cent. With comparable types of cases I have seen little reason to alter this view, and although some who heard and discussed that paper seemed to think the figure should be higher, I note that a recent report of the British Medical Research Council (that of Bentley, already cited) gives 10 per cent as the maximum that can be treated with advantage by this method. Reasons why American institutional workers incline toward much higher figures may be the tendency to earlier use of pneumothorax in the United States and its greater use in cases with limited lesions, sometimes asymptomatic, sometimes even without positive sputum, and less strictness as to contraindications, for example, use in cases with extensive bilateral lesions, either as a last resort or with the definite intention of inducing and carrying on bilateral pneumothorax.

How early should pneumothorax therapy be begun? As we have just indicated, there is a marked recent tendency to apply this procedure in cases with less and less involvement and to begin it even sooner and sooner, and with less preliminary observation. I am not sure that this is entirely warranted by actual consideration of facts and figures. In older children and adolescents with parenchymal lesions of the adult type, who ordinarily do very badly under more conservative treatment, as shown by Morgan at Westfield and others, immediate induction of pneumothorax is probably warranted and justifiable in a large proportion of cases, although I think we are not yet in a position to be too sure of our ultimate results, and exacerbations and contralateral extensions seem very frequent in this group. I do think such precipitancy is not warranted in adults, however, unless the lesion is demonstrably progressive and destructive in type, and this can hardly be determined in cases of recent onset without *a reasonable period* of observation under favorable conditions. We are often given pause by the rather frequent occurrence of marked clearing of lesions and sometimes even closure of cavities under comparatively brief periods of rest and sanatorium routine. Considering this, can we be justified in wholesale induction of pneumothorax, knowing that it must be continued in most cases for not less than two or three years and that pleural complications are not uncommon, even in comparatively early cases?

In what proportion of patients treated by pneumothorax may an effective collapse be expected? The answer to this will vary considerably with the material, that is, the proportion of advanced, long-standing and serious cases included, and also with the strictness of the criteria of what constitutes an effective collapse. The Committee on Artificial Pneumothorax of the American Sanatorium Association used three principal criteria, but did not strictly define what it meant by "sputum-negative." At present there are many who do not consider a collapse effective if any considerable bands of adhesions remain attached near involved portions of the lung, even though cavities appear to be closed. Even with the less strict criteria it is a minority of the general run of cases that achieves an effective collapse, and while the proportion can be increased by pneumonolysis in selected cases, many adhesions cannot be so done away with. There is a trend toward too great popularization of the method, minimizing the disadvantages and failures. I have in mind a motion-picture film sponsored by the New York or the National Tuberculosis Association publicity service; it shows Aunt Emma, who has had pulmonary tuberculosis for twenty years or more, having her lung collapsed apparently instantaneously, a large cavity disappearing as if by magic. This is certainly misleading, for we know that we should be unlikely to obtain any pneumothorax at all in such a case, and, if we did, it is almost certain that we could not successfully collapse Aunt Emma's cavity.

How long must pneumothorax treatment be continued? I think the answer depends on two principal factors: the duration of effective collapse, and the character and severity of the original lesion. Many patients can probably be discharged with reasonable safety after what may be considered an adequate stay at a sanatorium and be continued as ambulant cases, but if this is done, close and careful observation and supervision must be had. Too many ambulant patients receive very poor treatment and very careless supervision.

Finally, I think we should consider more carefully, not only the *type of lesion* for which collapse therapy is proposed, but the *optimum time* to apply any given procedure. This optimum time will be earlier for pneumothorax, of course, than for thoracoplasty, yet it is probable that certain *very acute cases* are not responsive to any type of collapse therapy. Ornstein believes this has been proved with acute pneumonic phthisis, pleuropulmonary perforation occurring in 40 per cent of the cases if they are given pneumothorax before cavitation is demonstrable and localized. The very acute group in the Loomis Sanatorium studies also showed the poorest response. Serious complications and progression of disease usually occur in this group, even under pneumothorax.

These remarks are not intended to belittle the excellent results that have been achieved with collapse therapy, but rather to indicate the need of a critical attitude of mind.

DR. GERARDO M. BALBONI, Boston: Every case of pulmonary tuberculosis is a candidate for collapse therapy. My experience goes back to 1909, and I believe I treated the first case of incipient pulmonary tuberculosis by artificial pneumothorax. The lesion was in the upper right lobe, and the diagnosis was made by x-ray by Dr. George Holmes. This patient was kept under observation for seven months. During this time she married, and since then has had numerous children and has kept in good health.

I believe that pneumothorax should be done early in the open active cases. It is the quickest method for starting on the road to recovery, shortens sanatorium stay, and enables the patient to begin active work sooner. The

lung should be so collapsed as to close cavities and stop cough and sputum. Frequent fluoroscopic examinations, as well as an occasional plate, should be made; and note that the lung should be maintained collapsed at practically the same level that maintains the cavities closed. The better the collapse, the better will be the result. The best collapse by pneumothorax is obtained where a free pleural cavity exists. Where adhesions prevent a proper collapse the Jacobaeus operation or a phrenicotomy may be indicated. Where a pulmonary tuberculous lesion cannot be treated satisfactorily by the above methods, a thoracoplasty is indicated.

The shifting of the mediastinum should not cause undue alarm. The development of a pleural effusion is of more importance. Mediastinal hernias do occur in certain cases, but do not usually give rise to serious inconvenience per se.

How long should one keep up pneumothorax? This depends on the type of lesion. Roughly, the exudative lesions without cavitation require not less than a year; the chronic cases with cavitation may have to be kept collapsed four or more years. The question arises, Does the lung re-expand and function after having been kept collapsed for years? It does; but never re-expands to normal. In one of my cases kept collapsed for five years, the lung re-expanded about one third. The patient did more than ordinary work, but on exertion was somewhat dyspneic and cyanotic.

The rules to follow during pneumothorax therapy are as follows. Keep the patient in bed at rest until he is symptom-free and has a sense of well-being. Then allow him graduated exercise. Watch the pneumothorax closely. As the patient keeps free from symptoms and gains in general well-being, allow more activity until he is leading a regular life with moderate work. The case has now become ambulatory. Most patients will do remarkably well under these conditions, but vigilance on the part of both doctor and patient is essential for lasting results.

THE CHAIRMAN: I am sure we all like to hear from Dr. Balboni because he was one of the first in this part of the country to give pneumothorax. He told me once how he used to generate his own nitrogen on the kitchen stove when he first began giving pneumothorax treatments.

The paper by Dr. Wilson and the various discussions have opened up a great deal of interesting material, particularly on the more recent problem of the value of phrenic compression, especially in advanced disease. It was from John Alexander's clinic in Ann Arbor that enthusiasm for compression came. A short time ago when in Ann Arbor I was interested to find a growing conviction among members of the clinic that the result of their early phrenic compressions has not been so good as they had expected. There was a group of cases, of which they have now published the results, in which they had done temporary phrenic compressions at the very earliest time, when the diagnosis was made by x-ray much the same as in these nurses here, and before there were symptoms of the disease. It was discovered that many of those on whom this operation had been performed developed signs of active tuberculosis within one to two years. Consequently temporary phrenic compression in early tuberculosis is regarded of doubtful value.

Are there any further questions from the floor?

DR. JACOB KAMINSKY: Dr. Wilson's paper is a very interesting report of a large amount of work well done. In 1931, at a meeting of the Eastern Section of the American Sanatorium Association at Mount MacGregor, Dr. Wilson showed us an interesting case of bilateral pneumothorax complicated by repeated spontaneous pneumothoraxes. I expected to hear a larger number of bilateral pneumothorax cases mentioned in his report this morning.

I was particularly impressed with the fact that in his large series of patients treated with pneumothorax there were comparatively few tuberculous empyemas. This is a most troublesome and dangerous complication, and I should like Dr. Wilson to tell us in a little more detail how this complication has been handled. In his first case, just what was the sequence of events in the treatment? How did he clear the empyema? Was it necessary to aspirate between the thoracoplasty stages? Was the empyema cleared soon after the final operation? I should also like to ask Dr. Wilson whether he and his associates perform thoracoplasty in the presence of counterlateral pneumothorax.

THE CHAIRMAN: Are there any other questions before we give Dr. Wilson a chance to answer some of the various questions which have been asked him?

DR. WILSON: I heartily agree with Dr. Peters that the statistics on artificial pneumothorax and these other measures leave a good deal to be desired. I was quite surprised when I attempted to dig out these statistics. All of you who are actively engaged can appreciate how difficult it is to get these things out while trying to carry on a day's work. Drs. Alexander and Nehil published a paper on phrenicectomy, and the latter had eight months free to gather the statistics for the paper.

I think we are not in a position today to say definitely what the end results with artificial pneumothorax are. I took our cases collapsed prior to May, 1932,—five years ago,—and particularly studied the re-expanded cases, because no one is in a position to state anything about these cases until he has followed them for a long period of time. It is felt that five years is by no means long enough to give final results.

I do not know why English statistics are so poor as compared with the figures published in this country and others. They certainly do not show the percentage of recoveries and percentage of effective collapses that we obtain. Whether they treat much more advanced cases, or whether they do not push the pneumothorax early and thoroughly, I do not know. Maybe the climate is responsible.

As to the percentage of admissions that is suitable for pneumothorax, we feel pretty strongly that it should be thought of in almost every case of tuberculosis, but we do not agree with Dr. Balboni that it should be applied in every case. We see many far-advanced cases in which it cannot be applied. On the other hand, we see a number of students, nurses and other young individuals with closed tuberculosis who do very well on bed-rest and a certain amount of graded exercise. If the patient has a cavity, however, or x-ray evidence of progressing disease, and has a positive sputum,—not one positive sputum by concentration but repeated ones,—we almost never regret turning to pneumothorax or collapse in some form. If we do not turn to it, relying upon six months or a year of bed-rest, we later often regret that we did not consider pneumothorax at the time when we first saw the patient, as we lose a lot of time and the condition becomes worse. Such cases have been discharged as quiescent; we see them again in a year or two with a cavity and with positive sputum, and then have to turn to artificial pneumothorax.

I was very much surprised in going over our figures on phrenicectomy to see what good results we had obtained, particularly with medical students and nurses. It is said

that you should not expect more than about one in six, say 16 per cent, to get an effective collapse with phrenicectomy alone; yet, particularly with the moderately advanced and even far-advanced cases, an astonishingly high percentage showed a good result from permanent phrenicectomy.

I am against temporary phrenicectomy. Few cases will run into trouble later with high diaphragm. Tuberculosis is a long-drawn-out affair, and a temporary phrenicectomy is much too brief to produce the results desired. We have had temporary phrenicectomies repeated four or five times on the same patient. In one case after four such phrenicectomies we turned to artificial pneumothorax, which is what we should have done in the first place.

Another thing that surprised me in preparing this paper was this: I had always felt that the older figures showing a high mortality with cavity cases were quite misleading, but when I looked up Dr. Brown's report on the far-advanced cases I found that he had reported 76 per cent dying within five years, which is pretty close to the Barnes figure. Over 50 per cent of the moderately advanced cases die within five years. I heard this morning a report from the Middlesex Sanatorium showing a mortality of 79 per cent of the far-advanced cases. So that I think we can assume that a patient who has a cavity over 2 cm. in diameter, with a persistently positive sputum, has a pretty poor chance of surviving five or ten years. If such patients do survive, they are at best invalids and a distinct danger to themselves, their families and the community.

We have used the term "effective collapse," as defined by the Sanatorium Association, that is, closure of the cavity as seen in repeated x-rays of the chest, and a persistently negative sputum by concentration methods. If there is persistent sputum, and it is anything more than salivary sputum, we generally find positive sputum by concentration if we look long enough. It is not enough to do one concentration. That is particularly true of the cavity cases. A great many of these have persistently positive sputum as long as there is any sputum.

I do not agree with Dr. Balboni that every case should have artificial pneumothorax. I do agree that every case is a candidate and should be considered as such and given the benefit of early and thorough collapse if there is any indication.

Our results in cases of tuberculous empyema have been quite good. Great care must be used not to infect the chest wall. We have to keep the patient lying on the opposite side for a considerable period of time. If he does not develop sinus of the chest wall, eventually the pus is absorbed and a dry pleura results in almost all cases.

The patient reported as Case 1 had had fluid for about six months, reported purulent, and was treated by repeated aspiration with replacement by air. She had some pus in the pleural space up to the time of the thoracoplasty. Before the operation the fluid was entirely withdrawn and replaced with air. After the operation the air was withdrawn rapidly, under low negative pressure, with no subsequent reaccumulation of the fluid or symptoms. The space may be all sealed off; we do not know. But clinically the treatment has been successful.

We have not done artificial pneumothorax on the side opposite a thoracoplasty. In this case that I have cited we performed artificial pneumothorax on the side opposite to the original artificial pneumothorax for ten or eleven months before resorting to thoracoplasty. The x-ray and the negative sputum led us to take a chance and to re-expand that lung at the same time as the thoracoplasty.

We have done phrenicectomy on the side opposite the thoracoplasty twice. In one case the outcome was quite successful; in the other the patient died, although she had fairly good vital capacity beforehand. In the last case the phrenic nerve was injected with novocain; we waited at the operating table half an hour to see if the patient was in distress, but she was not. When the clamp was put on the phrenic nerve she became very short of breath, and died within twenty-four hours.

THE CHAIRMAN: It is the custom of the Section of Tuberculosis to elect officers for the section; these officers usually remain in office for a two-year period. Last year the present officers were elected: Olin S. Pettingill, chairman, and Theodore L. Badger, secretary.

Under these circumstances I ask someone to move that the present officers be continued in office, or to state what alternative is the desire of the section.

The motion for re-election was made, seconded and carried.

DR. HENRY D. CHADWICK, Boston: I was one of the proponents of this section when it was organized some years ago as a separate group of the Massachusetts Medical Society. I now feel that the section has ceased to be a useful unit of the Society, and for that reason I should like to get an expression of opinion from the group here as to whether it is worth while to go on. When this new section was proposed, we felt that it would give an opportunity for the general practitioner to attend the meetings and keep in touch with the progress made in the diagnosis and treatment of tuberculosis. That theory never worked out in practice, since very few outside of those engaged in tuberculosis work appeared to be interested in the programs presented. It seems to me that the Trudeau Society, which meets three or four times a year to discuss the various aspects of tuberculosis and presents clinical and technical papers, such as we have had today, covers this field. Then, again, there were some very interesting papers read this morning in other sections, which we should have heard. I believe that the time has come to consider abandoning this section, and I move, Mr. Chairman, in order to get this matter under discussion, that the Section of Tuberculosis be discontinued, and that that recommendation be made to the officers of the Massachusetts Medical Society.

The motion was seconded.

THE CHAIRMAN: I am much interested to hear Dr. Chadwick speak in the way he has. I was about to bring up that very subject. We have in Boston the Trudeau Society, which is not made nearly so much of as it might be. I agree with everything Dr. Chadwick has said. There are a great many people interested in tuberculosis and its allied diseases who are not members of the Trudeau Society, and who could well be gathered together for a discussion of technical matters and matters of interest to those who are interested in tuberculosis. I do feel that papers on tuberculosis or pulmonary diseases should be made of interest to the general practitioner who never comes to our meetings.

The question was put to a vote and carried.

THE CHAIRMAN: I think we should consider that the meeting is adjourned.

ASYMPTOMATIC SCURVY

Its Relation to Wound Healing and Its Incidence in Patients With Peptic Ulcer

THEODORE H. INGALLS, M.D.* AND HARRY A. WARREN, M.D.†

BOSTON

OUR purpose in this report is to present a series of 20 cases of peptic ulcer with studies of the vitamin C level in the blood plasma of these patients; to point out the relation of the low values found to the deficient vitamin C intake; and to discuss the possible effects of asymptomatic scurvy on healing of the ulcer, on gastrointestinal hemorrhage and on wound healing following surgery on such patients.

In an independent approach to this problem, Archer and Graham[1] state that latent scurvy is frequently a factor in the death of patients following operation on the stomach. It occurred to these authors that, owing to the long period of ill health and the prolonged dieting to which these patients were submitted, they might be in the sub-scurvy state. They state: "It is well known that the wounds of patients with scurvy heal badly, and Hess says that 'they assume a hemorrhagic aspect, the edges becoming bluish or livid and showing no tendency to heal.' " The histologic basis for this phenomenon has been demonstrated by Wolbach and Howe[2] to lie in the "inability of the supporting tissues to produce and maintain intercellular substance." Archer and Graham report that the cause of death of 51 patients after operation on the stomach at St. Bartholomew Hospital during the past sixteen years was peritonitis in 16 cases. In 12 of these patients "the peritoneal infections appeared to have arisen as a bacterial leakage at the site of anastomosis, following an almost complete absence of fibrinous response along the suture line."

Although frank scurvy is of relatively rare occurrence, Lanman and Ingalls[3] have shown that "guinea pigs maintained on approximately one fifth of the minimal preventive dose of ascorbic acid exhibited healing of operative incisions inferior to normal controls." They further stated: "It has been proved histologically and chemically that asymptomatic scurvy is far more common in infants and children than has been realized. Its existence in older patients, though not yet proved, undoubtedly occurs." They concluded that "a partial vitamin C deficiency is of more importance in the healing of surgical wounds in human beings than has hitherto been appreciated."

A case similar to those of Archer and Graham is that of B. L., a fifty-four-year-old Jewish laborer, who entered the Peter Bent Brigham Hospital complaining of epigastric distress. On surgical exploration he was found to have an early carcinoma of the pylorus. He had been following a diet for over six months consisting of milk and cream, three to four eggs a week, and no other food. At operation the carcinoma was removed, and a gastrojejunostomy performed. Six days after the operation the abdominal wound broke open. He died two days following resuture of the wound. At autopsy the surgical incision was open throughout its entire extent. The edges of the wound were greatly thickened, edematous and necrotic. The skin for approximately 4 cm. on either side showed a deep purplish black discoloration. The subcutaneous tissues were stained dark greenish black and were extremely soft and friable. There was considerable exudate, though no frank pus was seen. The gastrojejunostomy and the sutured portion of the duodenum had also broken open, and there was a definite peritonitis.

It is interesting to compare the description of the operative wound as objectively described by the pathologist with the description already quoted from Hess, and also with Lanman and Ingall's[3] note on wounds experimentally produced in guinea pigs—"the scar tissue of the wounds of the normal animals after rupture was pink and firm, while that of the wounds of the scorbutic animals was livid and of soft consistency."

In view of Archer and Graham's studies and the work of Lanman and Ingalls, it would seem that this patient was in a scorbutic state which may have been the reason the surgical wounds of the abdomen, gastrojejunostomy and duodenum all gave way.

It seemed to us profitable to look for evidence of asymptomatic scurvy in other patients with chronic gastrointestinal disorders. Archer and Graham[1] stated that "many patients who have a gastric or duodenal ulcer and have been treated with a strict diet, lack ascorbic acid in their bodies." These workers performed urinary studies to detect the degree of saturation of 9 patients on a gastrointestinal diet. They found 3 of these patients were "less saturated than the control," but that their urinary excretion returns "were better than those of a patient with mild scurvy." However, it has been pointed out by Ingalls[4] that saturation "is a state so far removed from scurvy that urinary

From the medical service of The Peter Bent Brigham Hospital, Boston.
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tests become of questionable value as a purely diagnostic procedure." He found plasma determinations to be the best single index of vitamin C nutrition in infants, and the correlation of plasma values with the nutritional state could be represented as follows:

State of Vitamin C Nutrition	Plasma Ascorbic Acid
	mg. per cent
Optimum	
Saturation	2.00—1.00
Normal	1.00—0.70
Low normal	0.70—0.50
Suboptimum	0.50—0.30
Deficiency	
Asymptomatic scurvy	0.30—0.15
Clinical scurvy	0.15—0.00

These figures can undoubtedly be extended to adult investigations, and Pijoan and Klemperer[5] at the Peter Bent Brigham Hospital, while studying the accuracy of methods used to assay the ascorbic acid of the blood, "obtained values varying from 0.65 to 2.00 mg. per cent in the blood plasma of one hundred and fifty normal individuals."

PRESENTATION OF DATA

Because of the foregoing facts, determinations of the amount of vitamin C in the blood plasma have been made on 20 patients with peptic ulcer seen in the gastrointestinal clinic at the Peter Bent Brigham Hospital, and on 2 normal controls. In

TABLE 1. *Summary of Dietary and Other Data and the Plasma Vitamin C Content in All Cases.*

Case No.	Initials	Sex	Age	Diagnosis	Approximate Time on Bland Diet	Days Per Week With an Orange or Tomato Juice	Plasma Vitamin C*
							mg. per cent
1	P.G.	Male	41	Duodenal ulcer	18 mo.	3	0.10
2	M.K.	Female	51	Gastric ulcer, hourglass stomach	4 mo.	Irregularly	0.00
3	I.L.	Female	35	Duodenal ulcer	3 mo.	None	0.20
4	H.L.	Female	66	Gastric ulcer	6 yr.	Irregularly	0.20
5	I.R.	Female	26	Duodenal ulcer	7 yr.	4	0.10
6	T.K.	Male	29	Duodenal ulcer	6 mo.	Daily for 4 mo.	0.40
7	M.T.	Female	35	Duodenal ulcer	11 mo.	Irregularly for 3 mo.	0.10
8	C.E.	Male	53	Duodenal ulcer, gastrojejunostomy, gastrojejunal ulcer	7 yr.	3 for 2 yr.	0.10
9	M.R.	Male	64	Duodenal ulcer	2½ yr.	None	0.10
10	R.S.	Female	47	Duodenal ulcer	7 mo.	5 for 6 mo.	0.00
11	F.K.	Female	63	Duodenal ulcer	4 yr.	None	0.10
12	J.P.	Female	58	Gastric ulcer	11 mo.	Daily	1.15
13	J.P.P.	Male	30	Duodenal ulcer	3 mo.	Daily	1.15
14	H.S.	Male	23	Duodenal ulcer	10 mo.	None	0.20
15	C.McI.	Female	42	Duodenal ulcer	4 yr.	None	0.20
16	B.K.	Female	68	Gastric ulcer	1 yr.	None	0.20
17	N.C.	Female	52	Duodenal ulcer	3 yr.	None	0.30
18	O.S.	Male	56	Gastric ulcer, duodenal ulcer	10 yr.	None for 3 wk.	0.35
19	S.C.	Male	45	Duodenal ulcer	7 days	None	0.60
20	C.F.	Male	24	Congenital anomaly of duodenum	3 wk.	None	0.20
21	T.H.I.	Male		Normal control			1.50
22	H.A.W.	Male		Normal control			1.45

*Expressed to nearest .05 mg. above titration reading. Although the experimental error of the method is about 0.1 mg. per cent, it is present only on the minus side of the titration figure, owing to the nature of the end poin For instance, 0.10 mg. per cent might in reality be 0.05 mg. per cent, but could not be greater than the former figure.

all the determinations, the protein of the blood plasma was precipitated with metaphosphoric acid, and all samples were preserved with potassium cyanide.[4, 5]

These patients had been treated for their disease for varying periods with one form or another of bland diet. Some had been following the "first-week Sippy diet," which consists of hourly feedings of milk and cream and six small feedings of either cereal or eggs. Others had been following the "fourth-week Sippy diet," which, in addition to the hourly feedings of cream and milk, allows three small meals of meat, potatoes, puréed vegetables and stewed fruit. All the patients on the latter diet were allowed tomatoes and were advised to take orange juice once daily.

In Table 1 it is seen that the two controls had a normal amount of vitamin C in their blood plasmas, namely, 1.5 and 1.45 mg. per cent. Of the 20 patients with an ulcer, 18, or 90 per cent, had less than the normal amount of vitamin C in the blood. This ranged from zero to 0.6 mg. per cent of blood, with an average of 0.19 mg. per cent. Only two patients, Nos. 12 and 13, showed a normal content, namely, 1.15 mg. per cent in each instance.

DISCUSSION

It is evident that in this series a large proportion of the patients with peptic ulcer were markedly deficient in vitamin C. Several reasons for this come to mind:

1. That the patients were obtaining an inadequate amount of vitamin C.
2. That the vitamin C was being destroyed in the gastrointestinal tract before absorption could take place. It is known that alkalies tend to destroy this substance, and all the patients were taking alkaline powders daily.
3. That there might have been something about these patients which prevented the normal absorption of this substance.

Theoretically, most of these patients should have been receiving a maintenance dose of the vitamin if they had taken the orange juice as advised. Careful questioning, however, revealed that many of the patients were not doing so either because they thought it was acid and as such should not be taken, because they did not like it, or because of some other reason.

It is of interest to study their dietary intake of vitamin C more in detail, comparing the length of time on a low intake with the amount of vitamin C in the blood. Ten of these patients had taken no fresh fruit regularly for periods ranging from three months to five years. Their blood vitamin C varied from 0.0 to 0.3 mg. per cent and averaged 0.16 mg. per cent. Three patients had been on first-week Sippy diets for periods from one to three weeks, preceded by a normal intake of vitamin C. Their vitamin C ranged from 0.2 to 0.6 mg. per cent and averaged 0.38 mg. per cent. Five patients had taken orange juice three to six times a week for periods from three months to two years. Their blood vitamin C varied from 0.00 to 0.4 mg. per cent and averaged 0.14 mg. per cent. However, in 4 of these cases the periods had been preceded by several years when no citrous fruits had been taken. The remaining case had been on a first-week Sippy diet for two- or three-week-periods some seven times in six years. The 2 patients with normal vitamin C values had taken citrous fruits frequently throughout their illness.

From the foregoing data it seems that one must conclude that at least one reason for the low values was an inadequate intake of vitamin C. Since these figures were obtained, 2 patients have been given massive doses of ascorbic acid, with a rapid rise in the blood content of that substance to normal. This would seem to suggest that these patients are capable of absorbing vitamin C in a normal way, and that alkaline therapy will not prevent a normal absorption if a sufficient amount of the vitamin is ingested. However, further work must be done along these lines.

These data bring up an interesting question as to how much importance the low vitamin C content in the blood may have. None of these patients showed evidence of clinical scurvy, and experience with the Sippy diet by a large number of observers has shown that the incidence of scurvy occurring in these individuals is very slight. On the other hand, 8 of our 20 patients had had gastrointestinal hemorrhages in the past. Six of these patients had a blood value of 0.2 mg. per cent or less, one of 0.35 mg. per cent, and one of 0.6 mg. per cent. However, the blood specimens were not taken at or shortly after the time of the hemorrhage, so that the exact level at the time the bleeding occurred is not known. Further studies on this aspect of the problem are being pursued.

The surgical implication which has already been brought up becomes accentuated when it is considered that such patients are not only often operated upon many weeks after the inauguration of a dietary regime, but are subsequently maintained on intravenous fluid or milk long enough to exhaust the small tissue reserve of vitamin that remains. Finally, although there is no indication that a lack of vitamin C is an etiologic factor in the genesis of duodenal ulcer, it is worth while to consider the role of the vitamin in the healing of the lesion when once formed.

SUMMARY AND CONCLUSIONS

The amount of ascorbic acid in the blood plasma has been determined on 20 patients with gastric or duodenal ulcers. Eighteen, or 90 per cent, of them were found to have low values, 12 of whom had values of 0.2 mg. per cent or less as compared with the normal range of 0.6 to 2.0 mg. per cent. In most of these cases it could be demonstrated that the patients had actually taken an inadequate amount of vitamin C, and there is evidence that if a sufficient amount of the vitamin is ingested the blood values can be raised to normal. The theoretical importance of these findings in relation to the healing of peptic ulcer, to the bleeding of the ulcer and to surgical intervention is discussed.

It is important for the clinician to make sure that patients with peptic ulcer are receiving an adequate amount of vitamin C.

We are indebted to Dr. E. S. Emery, Jr., for his help in conducting this study.

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RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR JULY, 1937

Diseases	July 1937	July 1936	Five-Year Average*
Anterior Poliomyelitis	30	3	30
Chickenpox	439	312	367
Diphtheria	26	36	62
Dog Bite	1202	1199	1040
German Measles	59	101	174
Gonorrhea	506	548	577
Lobar Pneumonia	183	214	148
Measles	673	1282	1041
Meningococcus Meningitis	5	6	6
Mumps	171	445	288
Scarlet Fever	263	257	365
Syphilis	514	476	361
Tuberculosis, Pulmonary	412	301	328
Tuberculosis, Other Forms	37	41	42
Typhoid Fever	15	43	24
Undulant Fever	4	4	2
Whooping Cough	789	467	595

*Based on figures for preceding five years.

RARE DISEASES

Anterior poliomyelitis was reported from Adams, 1; Boston, 6; Brockton, 1; Chelsea, 1; Everett, 1; Fall River, 1; Greenfield, 1; Haverhill, 2; Ipswich, 2; Lynn, 1; Manchester, 1; Newburyport, 4; North Adams, 2; Quincy. 1; Revere, 1; Rowley, 1; Salisbury, 1; Somerville, 1; Williamstown, 1; total, 30.

Anthrax was reported from Lynn, 1.

Diphtheria was reported from Boston, 2; Chelsea, 1; Fall River, 4; Lancaster, 2; Lynn, 3; New Bedford, 1; Newton, 1; Palmer, 1; Peabody, 4; Somerset, 1; Tewksbury State Infirmary, 5; Westboro, 1; total, 26.

Dysentery, bacillary, was reported from Fall River, 1; Milford, 1; Worcester, 1; total, 3.

Encephalitis lethargica was reported from Springfield, 1; Worcester, 1; total, 2.

Malaria was reported from Avon, 1; Boston, 1; Pembroke, 1; total, 3.

Meningococcus meningitis was reported from Barnstable, 1; Boston, 1; Falmouth, 1; Methuen, 1; Montague, 1; total, 5.

Paratyphoid was reported from Adams, 1; Belmont, 1; Boston, 3; Brookline, 1; Cambridge, 1; Cohasset, 1; Deerfield, 4; Greenfield, 2; Lincoln, 1; Lowell, 2; Lynn, 1; Milton, 1; Northampton, 1; Quincy, 1; Stoneham, 2; Swampscott, 1; Wakefield, 1; Waltham, 1; Wellesley, 2; total, 28.

Septic sore throat was reported from Boston, 4; Braintree, 1; Brookline, 1; Lynn, 1; Medford, 1; Revere, 1; Watertown, 1; total, 10.

Tetanus was reported from Boston, 1; Taunton, 1; West Springfield, 1; Worthington, 1; total, 4.

Trachoma was reported from Watertown, 1.

Trichinosis was reported from Boston, 1.

Typhoid fever was reported from Fall River, 1; Holden, 1; Lynn, 1; Longmeadow, 1; Medford, 2; Melrose, 1; New Bedford, 1; Newburyport, 1; North Brookfield, 1; Quincy, 2; Randolph, 1; Williamstown, 1; total, 14.

Undulant fever was reported from Ashby, 1; Hopkinton, 1; Northampton, 1; Springfield, 1; total, 4.

Pfeiffer Bacillus meningitis was reported from Longmeadow, 1.

Diphtheria continued to show a record low incidence for the tenth consecutive month.

For the first time this year the monthly incidence of meningococcus meningitis fell below the five-year average.

The reported incidences of mumps, measles, German measles and scarlet fever continued below the five-year average.

The reported incidence of typhoid fever continued below the five-year average, with record low figures for any year to date.

Paratyphoid fever and chickenpox had the highest reported incidences both for July and any year to date.

The reported incidence of whooping cough continued above the five-year average, with record high figures for any year to date.

Lobar pneumonia, pulmonary tuberculosis and undulant fever were reported above the five-year average for July.

The reported incidences of tuberculosis (other forms) and anterior poliomyelitis were not remarkable.

This month had the highest July incidence of animal rabies ever reported except for 1934. During July new foci for this year appeared in Douglas, Reading, Stoughton and Weston. There is an unusually wide distribution of animal rabies for the year to date.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 23361

PRESENTATION OF CASE

A forty-nine-year-old American college executive was admitted complaining of the vomiting of blood.

For about two or three years the patient had noted occasional burning sensations in the lower midabdomen. These were usually of short duration and occasionally associated with sensations of pressure in the same region. The discomfort usually occurred during moments of emotional stress but had no relation to meals and was not accompanied by other symptoms of significance. It was never sufficiently severe to cause him to seek medical aid. About seven weeks before entry he became conscious of much more severe, burning pain in the same region. The discomfort was continuous but not associated with nausea or vomiting and did not interfere with his sleep. It was not relieved by the ingestion of food. Bowel movements, which had previously been normal, were unchanged. About six weeks before coming to the hospital, however, he passed a tarry stool, details concerning which were not recorded. He continued with his usual activities and noted that the pain had subsided. During the day, however, there was slight dyspnea on moderate exertion. Shortly before returning home he passed another tarry stool, but did not feel particularly weak. He was awakened during the night by a desire to urinate. While in the lavatory he became nauseated, vomited a large amount of bright red blood, and lost consciousness. No details were noted relative to the period of unconsciousness, but subsequently he was brought to another hospital, where he received three transfusions during the first three days. His diet was rigidly limited, and gradually increasing amounts of food were permitted. He was discharged after four weeks. X-rays taken while in the hospital were said to show an ulcer. For two weeks before entering this hospital he remained at home, eating frequent small meals consisting of bland foods and taking Sippy powders. During this period he was symptom-free, but the stools were still black in appearance. There was a loss of 15 pounds in weight during the seven weeks preceding entry.

Physical examination showed a well-developed and nourished man in no acute discomfort. There was no obvious pallor. The heart was normal. The blood pressure was 125/70. The lungs were clear. The remainder of the examination was entirely negative.

The temperature, pulse and respirations were normal.

Examination of the urine was negative. The blood showed a red cell count of 4,700,000, with a hemoglobin of 80 per cent. The white cell count was 9,900, 80 per cent polymorphonuclears. A blood Hinton test was negative. A stool examination showed a negative reaction to the guaiac test. A gastric analysis showed a free hydrochloric acid of 51 units at the end of an hour. Guaiac tests on the gastric contents were negative.

A gastrointestinal series showed a smooth oval tumor, measuring 7 by 5 cm., on the anterior wall of the stomach. The tumor was attached by a broad base, about 6 cm. in length, and the upper margin was 5 cm. beneath the esophageal orifice. The surface of the tumor was smooth, except for a 1 cm. ulceration slightly above its center (Fig 1).

FIG. 1. X-ray showing tumor mass and ulceration.

The remainder of the stomach, the pyloric valve and the duodenum were negative. At six hours the stomach was empty, and the motor meal filled the colon fairly well. There was no evidence of splenic or hepatic enlargement.

On the third hospital day a laparotomy was performed.

X-RAY INTERPRETATION

DR. AUBREY O. HAMPTON: The examination shows a very obvious tumor on the anterior wall of the stomach which we could see before we gave the patient barium. The air in the stomach outlines it very nicely. It is a smooth, round tumor

with a very broad base and no tendency toward pedunculation. It appears as much an extrinsic as an intrinsic mass, and there is one point—at the site of a small deep ulcer in the superior margin—where you can see that the mucosa is definitely destroyed. You can see it here, filled with air. It is nearly 3 cm. deep. These two films would make it appear as though the tumor were much more irregular than all the other films. Here it appears as though there were fissures crossing it, but in none of the films does it appear nodular. It is very smooth. It is high on the anterior margin of the stomach. We have seen these same x-ray findings several times, and it has been reported here at these clinics as a very specific type of tumor.

Differential Diagnosis

Dr. Walter E. Garrey: Let us summarize this man's symptoms. He is forty-nine years old and has had slight gastric symptoms for two years. More severe, steady epigastric pain began seven weeks before entry. Finally, six weeks before entry he developed gastrointestinal bleeding which rapidly culminated in a massive hematemesis.

At entry here his physical examination was not remarkable. He was not anemic and was not bleeding, and at the moment he was symptom free.

We are told that his x-rays showed one lesion in his stomach, a smooth, oval tumor, 7 by 5 cm., with a broad 6 cm. base. This was attached to the anterior wall with the upper margin 5 cm. below the esophageal orifice. There was an ulcer crater, 1 cm. across, slightly above the center of the tumor.

I believe there is no need to look for any lesion other than this tumor to explain this man's symptoms. There was an ulceration in the tumor adequate to explain the recent increase in pain and the massive bleeding.

Our problem then is to consider the differential diagnosis of a solid tumor of the stomach with a small ulceration on its surface. In order of frequency we must consider (1) gastric carcinoma, (2) benign tumor and (3) sarcoma.

Gastric carcinoma is about two hundred times more frequent than either benign tumor or sarcoma. Pedunculated carcinomas do occur. However, the surface of a carcinoma should not be smooth. The area of ulceration should be larger. Such gross hemorrhage and remittent bleeding is unusual in a carcinoma, which usually causes slow, constant bleeding. The symptoms have been mild and not progressive. The patient was apparently in a good nutritional state. All these facts point strongly against carcinoma, and I do not believe that this tumor was a gastric cancer.

By similar tokens there is much in favor of this being a benign tumor. The clinical differentiation of benign gastric neoplasm is beset with many difficulties. Moreover, the various types of benign tumors are not clearly distinguishable by symptoms and signs. However, there are certain deductions that we may make. About half of the benign lesions are leiomyomas. The next commonest lesion is polyp. Angiomas, adenomas and dermoid cysts of the stomach are all very rare, each constituting about 10 per cent of the benign lesions. Although polyps have been reported up to 8 cm. in diameter, they are almost invariably associated with a stomach in which there is achlorhydria and often associated with pernicious anemia. This patient had a free hydrochloric acid of 51 units. I believe polyp is unlikely. It is very characteristic of all types of benign tumors that they often cause sudden, massive gastric hemorrhage. This is particularly true of the myomas. The average age at which they are found coincides with the age of this patient. All these benign tumors usually give a history somewhat suggestive of a mild peptic ulcer. About one fourth of the myomas have an ulcer crater on the surface. This tumor is, however, a little larger than is usual for any of the benign tumors.

Thirdly, if we consider sarcoma of the stomach, this is even less common than benign tumor. It is commoner in a younger age group. The duration and type of pain, the size of the tumor, the erosion and the massive hematemesis in this case, all are perfectly compatible with sarcoma. The larger size of the tumor is in favor of it. Lymphosarcoma is much commoner than leiomyosarcoma or fibrosarcoma and is apt to be more irregular and diffuse than the roentgenologist's description of this tumor. A smooth, solid tumor of this sort should not be lymphoma.

In conclusion then, I believe that the evidence is against this being carcinoma. I do not believe we can rule out a sarcoma, especially leiomyosarcoma. I do believe that we can rule out polyp. I think the weight of evidence is in favor of a benign gastric neoplasm, and more particularly of leiomyoma. I believe that the crater-like ulcer on the tumor explains the seriousness of the hemorrhage and the pain.

Dr. Tracy B. Mallory: Dr. Garrey has approached his differential diagnosis according to the more or less classical treatment in the textbooks, but I question this method of attack. It seems to me, perhaps simply because of my histologic training, that to lump together as sarcoma such totally unrelated lesions as fibrosarcoma and leiomyosarcoma, on the one hand, and lymphosarcoma on the other, and to consider them altogether is rather illogical. I should rather have

someone try to rule in or out lymphoma, which is a distinct and very important entity in the stomach, and which has a very definite prognosis and corresponding line of treatment. The lymphomatous group should be contrasted with the group of spindle-cell tumors, including myomas and fibromas and the corresponding sarcomas. The differential diagnosis of the spindle-cell tumors, whether benign or malignant, can be made only with the microscope and with some uncertainty even then, whereas between the two groups the differences are so sharp and important that it should ordinarily be possible to distinguish them.

I wonder if Dr. Hampton would be willing to comment on the possibility of lymphoma in regard to this patient.

Dr. Aubrey O. Hampton: I cannot conceive of its being lymphoma because lymphoma does not usually produce such a smooth, round mass. It is more a flat, dish-like lesion, as in carcinoma. We confuse it with carcinoma almost invariably, but rarely with benign tumor. This picture we have now seen five or six times,—twice in the duodenum and about four times in the stomach that I can remember,—and only once has the lesion turned out to be a degenerated adenomatous polyp. All the rest of them have been either neurofibroma or myoma, and I have noted recently in the literature one other case, which makes seven in all.

Dr. Mallory: Dr. Hampton has brought out the point that I hoped he would, that is, on the x-ray appearance it is usually fairly easy to distinguish between the spindle-cell and the lymphoma groups.

Clinical Diagnosis

Leiomyosarcoma of the stomach.

Dr. Walter E. Garrey's Diagnosis

Leiomyoma of the stomach with ulceration.

Anatomic Diagnosis

Leiomyoma of the stomach.

Pathological Discussion

Dr. Tracy B. Mallory: The tumor in this patient was a leiomyoma as Dr. Garrey correctly predicted. It was a more or less cylindrical tumor, quite well encapsulated. On its surface it had a rather deep ulcer (Fig. 2). In the pathologic specimens these ulcers never look so deep as the x-ray people tell us they are. We did not make it quite a centimeter, but consider that a deep ulcer. Dr. Hampton said something about 3 cm. We are in no position to refute the roentgenologist's statement, however, because Dr. Hampton was seeing it with the mucosa filled with blood, and the mucosa is a very vascular organ. An ulcer unquestionably has a much greater thickness in life than it ever has at autopsy, and consequently these ulcerations appear larger at x-ray examination than they do when we get them in surgical specimens or at the autopsy table.

The tumor was growing with a moderate rate of

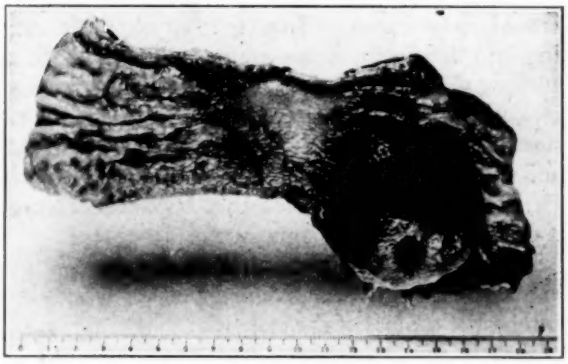

FIG. 2. Surgical specimen showing the leiomyoma and the ulceration.

speed, much faster than the average uterine fibroid, which is exactly the same type of tumor from the histologic point of view. Here and there in the sections we can find mitotic figures, but no other signs suggestive of malignancy. I think the likelihood of metastasis is extremely remote, and there is every chance that operation will result in permanent cure.

In the literature one gets the impression that almost all spindle-cell tumors of the stomach are myomas. I think there is no doubt that the majority of them are, but I have run across several cases over which there can be no doubt that we are dealing with neurofibromas, in other words the tumors seen in von Recklinghausen's disease, and I am rather inclined to think that a significant proportion of the many rather casually reported myomas of the stomach may have been fibromas or neurofibromas.

A Physician: Do you think there is much difference between the two clinically?

Dr. Mallory: No, the only difference is that neurofibromas tend to be multiple. I remember one case in another Boston hospital diagnosed in the pathology department as leiomyoma of the stomach that returned to the hospital three years later with a tumor of a dorsal nerve root in the spinal canal. I thought personally the gastric tumor looked more like neurofibroma than leiomyoma, but was not unprejudiced since I knew the whole story. A lot of these cases show ulcers overlying the tumor, and probably many of them never produce any symptoms until the ulcer develops. In some instances the ulcer is nothing but an ordinary peptic ulcer on the surface of the

tumor. In other cases I think the ulcer forms in an entirely different way. We had a case recently in the Phillips House which was quite interesting as an illustration of this mechanism. The mucosa had not yet ulcerated, but the entire central core of the tumor was necrotic. It looked as though a week more could hardly have passed before that bridge of mucosa must have succumbed, and one would have found a deep, shaggy ulcer penetrating into the center of the tumor. In some of these tumors the x-ray appearance is rather suggestive of such a pathogenesis. One can see the crater starting down into the tumor with a narrow mouth and then beginning at a deeper point to branch. Dr. Robert Vance tells me that he considers such a picture practically diagnostic.

Dr. Alfred O. Ludwig: What are the figures in your series of the relative incidence of lymphoma among the tumors of the stomach? My impression is that lymphoma is next most common to carcinoma.

Dr. Edward A. Gall: We have twelve lymphomas of the stomach.

Dr. Mallory: I am sure we must have fifteen or eighteen spindle-cell tumors.

A Physician: Is lymphoma more common than malignant, spindle-cell tumors?

Dr. Mallory: Yes, only three of the spindle-cell tumors have been frankly malignant.

CASE 23362

Presentation of Case

First Admission. A fifty-three-year-old American laborer was admitted complaining of a draining sinus in the stump of his right leg.

Seventeen years before entry the patient had had an amputation of his right leg about 10 cm. below the knee joint because of chronic osteomyelitis. He was known to have syphilis at that time. The glans penis had been amputated years before for an apparent chancre. Subsequently he carried on adequately with the aid of an artificial leg and remained well until fifteen months before admission. At that time he noted the discharge of varying amounts of foul-smelling, purulent material from the amputation stump. This continued until three and a half months before coming to the hospital, when the discharge became copious in amount and the posterior aspect of the stump became tender, reddened and edematous. Flexion of the knee joint became limited, and two months later an x-ray film showed very marked bone reaction in the stump of the tibia. Shortly before entry another x-ray showed two or three small loose fragments of bone near the lateral aspect of the tibial stump. These had not been present previously. The shaft of the bone showed decalcification without evidence of actual bone destruction.

Physical examination showed a poorly developed and nourished man, complaining of pain in the stump of his right leg. The tongue was dry and beefy red in appearance. The pupils were equal but reacted sluggishly to light. A few discrete shotty lymph nodes were present in the neck, axillae and groins. The heart was normal. The lungs were clear. The abdomen was negative. The right leg was amputated 10 cm. below the knee joint. The end of the stump was reddened, indurated and contained a small sinus tract from which a foul-smelling, thin, watery discharge drained. There was evidence of an old amputation of the glans penis.

The temperature was 102°F., the pulse 90. The respirations were 25.

Examination of the urine was negative. The white cell count of the blood was 13,600, and the hemoglobin 80 per cent. The nonprotein nitrogen of the blood was 21 mg. per cent. A blood Hinton test was positive, but the Wassermann test was negative.

On the third hospital day a biopsy of the sinus tract was done. Histologic section of this tissue showed epidermoid carcinoma, grade II. A week later a low thigh guillotine amputation was done. The temperature, which had fluctuated up to 102.5°F. prior to this time, rapidly returned to normal. The operative wound showed evidence of ready healing, and the patient was discharged on the twenty-seventh hospital day.

Final Admission (two months later). Following discharge the wound continued to heal. About a month before returning to the hospital the patient began to have pain and tenderness in the region of the sternum. This was not associated with cough or other significant symptoms. About a week later a nonpainful swelling appeared at the site of amputation and gradually increased in size. At about the same time he noted "hard cords" on the medial aspect of the right thigh. There was failing appetite, constipation and moderate malaise.

Physical examination showed no striking general change from that noted on the previous entry. The enlargement of lymph nodes previously noted was still present but the left axillary nodes were said to be somewhat larger. Along the medial aspect of the midportion of the amputation stump was a hard, fixed, irregular mass, the size of which was not recorded. Posterior to the right of the operative wound was a 4 by 3 cm., red, swollen, nontender mass, which produced a small amount of whitish watery material on pressure. A tender area was elicited over the midsternum and another

over the lateral aspect of the third right rib. The lungs were clear. The heart was not enlarged, but the sounds were faint in character. A harsh systolic murmur was audible at the apex. The blood pressure was 115/70. The abdomen was scaphoid, and there was increased resistance in the right upper quadrant. The liver dulness was percussed 3 cm. beneath the costal margin, but the edge was not felt.

The temperature was 100°F., the pulse 90. The respirations were 20.

Examination of the urine was negative. The blood showed a red cell count of 4,500,000, with a hemoglobin of 80 per cent. The white cell count was 10,300, 75 per cent polymorphonuclears.

An x-ray of the chest showed that both apices were less radiant than normal. There was evidence of thickening of the apical pleura and some mottling of the extreme apices on both sides. The lung fields were large and bright, and the diaphragm was low but moved fairly well with respiration. The heart shadow was not remarkable. There were no mediastinal masses. A film of the right femur showed some periosteal new-bone formation on the medial aspect at a small distance from the surface of the stump. There was no evidence of osteomyelitis.

On the third hospital day 15 cm. of the remaining stump was amputated, and a radical groin dissection performed. Histologic study showed recurrent epidermoid carcinoma, grade II, with metastases in the lymph nodes in the region of the tumor but none in the nodes removed from the groin. Following this operation the patient's temperature continued to fluctuate irregularly up to 102.5°F., and he complained of vague soreness in the center of his chest. There was cough productive of moderate amounts of yellowish sputum. From time to time small amounts of pus and necrotic material were removed from the operative wound. Three weeks after the last operation the patient began to complain of dull epigastric pain, and on the twenty-third hospital day began to have rather severe, intermittent, generalized abdominal pain. On the following day 50 ounces of fluid were removed from his stomach. The fluid gave a 2+ reaction to the guaiac test. The abdomen was diffusely tender but not spastic. Peristaltic sounds were not heard, and an enema produced no return. The white cell count was 55,000, 95 per cent polymorphonuclears. There was progressive abdominal distention. A laparotomy was performed.

Differential Diagnosis

Dr. Edward L. Young: A treatment of acute osteomyelitis so efficient that it will not leave behind a certain percentage of chronic osteomyelitis has yet to be devised. The treatment of the acute condition is still not settled, but we certainly are swinging toward conservatism at the present time. Nevertheless, given a case which does become chronic, a complete cure may never be reached in spite of vigorous treatment. In this case it was considered necessary to do an amputation as the method best calculated to give the patient relief. I do not believe the presence of syphilis had anything to do with the lack of healing. The course of the patient's life seemed to prove the adequacy of the treatment for he was free from trouble for nearly sixteen years. At that time he started to have some discharge from the stump. Following amputation of the leg, for any reason, it is not uncommon to have frequent minor ulcers develop in the stump, and sometimes there is a great deal of difficulty in keeping the wound closed.

In this particular case we have no evidence from the story that it was anything more than this until three and a half months before entrance, when there was evidence of trouble deeper in, as shown by the amount of discharge and the redness, swelling and tenderness in the stump itself. The x-ray also tells us that there was another attack of osteomyelitis in the bone. The patient himself was not in good shape on admission. He was dehydrated. A thin, foul-smelling discharge, whether from a sinus or from a cervix should always make us suspicious of malignancy. There is obvious sepsis as well, as shown by the temperature, the white count and the local appearance.

A biopsy was done which showed malignancy. This sinus had been present for only a little over a year, so that we have several hypotheses. Had this malignancy actually developed in about a year on the basis of chronic irritation? Had there been a chronic osteomyelitis in the stump for a much longer period without breaking loose? Or as an extremely wild possibility, did the original amputation actually leave behind a few carcinoma cells? Whatever the answer, it would seem as though adequate treatment was done in the guillotine amputation, as it removed the whole bone and neighboring joint. The wound held well, and everything seemed to be as it should, but it was only two months later when he returned.

Although we have no proof, there is the suggestion that his tumor had already metastasized to different parts of the body. Nevertheless, we have no right to deprive a person of any chance of life, however slight, and, accordingly, further amputation and dissection of Hunter's canal and the groin were done. It is interesting to know that in spite of the rapid recurrence of the carcinoma, which

was still graded as II, there was no evidence of metastases in the inguinal nodes. The continuation of chest symptoms makes us think still more of the probability of chest metastasis.

Then came the last act in the drama, which is puzzling to say the least—pain which was at first epigastric, then rather severe, intermittent, generalized abdominal pain, then evidence of paralytic ileus and peritonitis. What are the possibilities? It seems to me that with a phlebitis spreading into the pelvic veins, a peritonitis could arise from that source. Possibly a brand new condition intervened. If the epigastric pain had swung down to the right lower quadrant, I should say that he had acute appendicitis, but the picture is not definite enough to say that. The question is argued whether an idiopathic peritonitis ever comes from sepsis in a postoperative wound. I have never seen it, although I cannot deny that it might conceivably occur. He was operated on because they could not say that there was not a condition which would still give him a chance for life, but I have a feeling that a general peritonitis will be found without any cause explainable before the autopsy.

Clinical Diagnoses

Preoperative: Mesenteric thrombosis.
Epidermoid carcinoma of the leg.
Postoperative: Gangrene of the cecum.

Dr. Edward L. Young's Diagnoses

Epidermoid carcinoma of the leg with questionable metastases in the chest.
General peritonitis.

Anatomic Diagnoses

Operative wounds: old amputation of penis (for syphilitic chancre); amputation of right leg with groin dissection (epidermoid carcinoma of old osteomyelitis sinus); plication of cecum for gangrene.
Epidermoid carcinoma of leg with metastases to heart, lungs and adrenals.
Thrombosis of iliac and femoral vessels, right.
Pulmonary infarcts.
Pulmonary congestion and edema.
Pulmonary tuberculosis with cavitation, bilateral, healed.
Pleuritis, chronic fibrous, apical, bilateral.
Syphilitic aortitis.

Pathological Discussion

Dr. Benjamin Castleman: The surgeon's preoperative diagnosis before the laparotomy was mesenteric thrombosis, but what he found was a gangrenous pouch of the cecum. There was a strand of omentum adherent to the under surface of the scar of the previous groin dissection over the lower abdomen. The gangrenous cecum appeared as if it had been strangulated in the hernial opening. The uninvolved area was infolded and the abdomen closed. There was no peritonitis. Following operation his white count fell to 12,000, but the next day he went into collapse and died.

The autopsy showed numerous small metastases to the myocardium, the largest measuring about 3 cm. in diameter. Some of the nodules were growing in the papillary muscles and one in the interventricular septum. Each adrenal also contained metastases. What were believed grossly to be metastatic nodules in the lung proved histologically to be small infarcts, probably resulting from emboli arising from a thrombosis of the right femoral and iliac veins. There were, however, tumor cells in a few of the smaller pulmonary vessels. There was no large pulmonary embolus to account for his relatively sudden death, and it is quite possible that the cardiac metastases may have been responsible.

The cause of the gangrenous cecum was undoubtedly the constricting band of fibrous tissue. The vessels supplying the cecum were not thrombosed. The aorta showed some evidence of syphilis, characterized by lymphyocytic infiltration around the vasa vasorum of the adventitia.

The development of carcinoma upon a pre-existing chronic osteomyelitis is not too common. Dr. Edward Benedict* looked up the cases in this hospital about six years ago and found only 12 in 2,400 cases of osteomyelitis. In all his cases the lesions were in the leg, 8 of them in the tibia. The long interval between the initial lesion and the appearance of the carcinoma is quite characteristic. The unusual feature in this patient was the rapid metastatic spread. In none of our other cases was there any evidence of metastasis. It is possible, of course, that the cancer developed on the skin of the stump from the irritation of the prosthesis, but this is not so probable.

*Benedict, E. B.: Carcinoma in osteomyelitis. Surg., Gynec. & Obst. 53:1 (July) 1931.

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PROCRASTINATION

Procrastination, other than in the field of medicine at large, is a besetting sin of the doctor. This holds true even when he personally suffers from his own dilatoriness. No uninterested party objects to this, although it is always a source of astonishment to him. The doctor must therefore help himself. Such procrastination is especially prone to develop following the correction of either real or imagined acts of discrimination against the profession. It is, therefore, not surprising to find that only a small number of physicians or surgeons are taking advantage of the opportunity, recently afforded them, to increase the percentage of their collections in automobile injury cases.

The Special Committee on Hospital and Insurance Relations as designated by the Massachusetts Medical Society negotiated an agreement between the insurance companies, the hospitals and the physicians, the details of which were published in this *Journal* on March 25, 1937. Again in the issue of July 8 under the heading "Compensation in Automobile Accident Cases" they called the attention of the medical fraternity to the agreement and stated that forms whereby it could be consummated would be mailed shortly. On July 10 sample forms were mailed. A covering letter was included which gave the price of the forms and stated that members could obtain them from the secretary of the Society at 8 Fenway.

Now under the date of August 23 comes the following from Dr. Landesman, secretary of the special committee:

> The insurance companies have complained that many physicians are still unacquainted with our "agreement" and feel that we should do something in reference to enlightening members of the Society regarding this plan. This is rather odd for several announcements have appeared in the *Journal*, two sample blanks were sent to each member of the Society, and the plan is in full operation.
>
> Forms C and D decided upon by the Massachusetts Medical Society for use by physicians, samples of which have been sent to each member of the Society, are available at 20 cents per hundred. They were published by the Society to reduce the cost of printing to each member.
>
> Physicians are advised to send their orders in or call at the office of the Society at once.

The conclusions are inescapable. Either the members of the Massachusetts Medical Society do not want assistance in the collection of fees for their treatment of patients injured in automobile accident cases, or they are unable or unwilling to fill out the requested forms, or they are a group naïve enough to still believe that one can get something for nothing. The protest that resulted in the formation of the special committee that has this matter in charge and whose members have done an enormous amount of excellent work was to the effect that the members did want help in the collection of their fees. The forms, which, for legal reasons, must be followed if such help is expected are well within the intellectual and physical capacities of any member of the Society, and the work that we do should completely eliminate any last vestige of naïveté among us. We must then fall back at the end as we did at the beginning on our besetting sin of procrastination for the explanation of this delay in taking advantage of this opportunity that has been offered us. The cure is obvious. Write for these forms. *Write now* for these forms. *Do not procrastinate!*

ASYMPTOMATIC SCURVY

THE paper on "Asymptomatic Scurvy" by Doctors Ingalls and Warren appearing in this issue of the *Journal* excites in the editorially conditioned individual the cortical centers of memory and imagination which according to traditional teaching reside in the frontal lobes. To be sure, recent neurosurgical practice indicates that severing these lobes from the remainder of the brain results in a more normal behavior of the individual. This, however, does not concern the medical editorial writer except to warn him to avoid the neurosurgeon lest acquired normal cortical integration or sanity suggests ceasing editorial writing. But it serves to remind the editorial readers that abnormal and provocative reactions in their editorial servants are desirable and, if sometimes annoying, should nevertheless be viewed with tolerance. The alternative is an increase in editorials so prescribed by normal reactions as to lack any stimulus strong enough to excite the cortical centers of the reader.

Surely others than abnormal editors must react to the title, "Asymptomatic Scurvy." Since Hippocrates described cases of the disease two thousand years ago scurvy has been recognized by its signs and symptoms. Until 1926, when Wolbach and Howe by their studies in experimental animals identified the specific pathological lesion of the disease and Eliot and Park described the early x-ray manifestations of scurvy, the idea of asymptomatic scurvy was not entertained. Even then it remained merely as a possibility rather than a clinical reality, though the American fruitgrowers kept the concept continually before the public.

What then has led to the recognition of asymptomatic scurvy? Are the criteria for its detection well defined and easily obtainable? Does this indicate a significant advance in the early recognition of a curable disease or merely an ill-defined condition that will be held before the public by those who may derive benefit from so doing? Does the means of recognition suggest that earlier recognition of other diseases will be likely?

* * * * *

In 1928 Szent-Gyorgy isolated a highly reducing hexose derivative that in 1932 was identified as vitamin C and was called ascorbic acid. With this chemical knowledge of vitamin C, methods of determining its concentration in biological fluids were developed almost immediately and new criteria of estimating the deficiency state of patients were established. The first of these was concerned with estimating the so-called saturation of an individual by studying the urinary excretion of ascorbic acid while the acid was being ingested in specified amounts. Though standards according to this technic have been established, their reliability is questionable. Ill-defined suboptimal levels of vitamin C saturation appeared in the guise of scientific medicine as an explanation of many conditions. The sensitive receptors of medical editors flashed impulses that finally integrated into an unbridled vitamin C rampant on a background of a pyorrheic, rheumatic, nephritic, and purpuric public. The season was on for the fruitgrowers, but the buds were nipped by the chemical production of a cheap ascorbic acid tablet. Prospects looked bright as the medical charlatan viewed the field of loosely defined asymptomatic disease ripening for such legislation as embodied in Senator Lewis's bill whereby all doctors' bills not paid by the patient would be paid by the Government. But within the year reliable methods of determining the vitamin C concentration of plasma introduced the second new criterion for the detection and estimation of this deficiency state. The application of criteria established by the method has put asymptomatic scurvy on a sound basis, as illustrated by the paper by Ingalls and Warren.

* * * * *

Clearly asymptomatic disease should be diagnosed only on the basis of accurate evidence, if such early recognition of disease is to be a boon and not a burden to the public. Fortunately the determination by which asymptomatic scurvy may be diagnosed is a relatively simple one and should be widely used. The general method is obviously applicable to other diseases. Present work already indicates that the determination of the vitamin A concentration in plasma will replace the doubtful night-blindness criteria for the detection of vitamin A deficiency and thus another inadequately established condition will be more clearly defined.

MASSACHUSETTS MEDICAL SOCIETY

MEMORANDUM CONCERNING DIRECTORY

Section 3 of Chapter VI of the by-laws was amended at the annual meeting of the Society, held on June 3, 1937, to read as follows:

> Under the direction of the Committee on Publications he (the Secretary) shall issue at such intervals as may be determined by the Council a Directory of Officers and Fellows of the Society, which shall be furnished upon request to fellows who are not in arrears.

It will be necessary for the Council to take action at the October meeting and specify when the next directory shall be published.

A. S. BEGG, *Secretary.*

SECTION OF OBSTETRICS AND GYNECOLOGY

M. Fletcher Eades, M.D., *Secretary*,
19 Bay State Road,
Boston

Case History No. 36. Bleeding at Seven to Eight Months—Hydatidiform Mole

Mrs. M. H., a white woman, aged forty-six years, entered the hospital August 2, 1937, stating that twenty-four hours previously she had had a vaginal hemorrhage of an amount estimated by her to be 2½ quarts. She stated that blood covered the entire bathroom floor and that several heavy bath towels were soaked with bright red blood. The bleeding was painless and stopped entirely in ten minutes. She felt slightly weakened and went to bed. That afternoon she noted "pain in the groins" which was intermittent and "more like gas pains than labor pains." She thought that fetal movement had been more active since the bleeding. She entered the hospital twenty-eight hours after the hemorrhage.

No family history was obtained. The patient's health has always been good and she did not remember having had any childhood diseases. In 1931 she had a suspension, perineorrhaphy, trachelorrhaphy, anterior colporrhaphy and appendectomy, and the postoperative course was uneventful. In 1918, 1919 and 1921 she had normal pregnancies without complications terminating in normal deliveries. She had a breech delivery in 1923 and normal labors in 1924 and 1926, respectively. At five months gestation in 1925 she had a miscarriage, the cause of which was unknown. The menstrual periods have always been regular since twelve years of age and of the twenty-eight-day type. Her last normal period was December 15, 1936. On January 16, 1937, the patient started a flow of average menstrual amount which lasted for two days, and during February there was a slight flow on three different occasions. On May 1, when she applied for prenatal care, the significant findings were as follows: blood pressure, systolic 170, diastolic 90; pulse rate 135 per minute; urinalysis, very slight trace of albumin; blood hemoglobin 50 per cent (Sahli). The uterus at that time was consistent in size with a five months' pregnancy. The fetal heart tones were not heard.

On physical examination on admission the patient was thin, and there was a noticeable pallor of the face and mucous membranes. The teeth were poor, with many missing and carious. No cervical lymph nodes were palpated, and the thyroid was not grossly enlarged. The thorax was symmetrical with equal expansion. The lungs were resonant to percussion, and the breath sounds were normal. The heart rate was rapid, 130 per minute, and no gross enlargement of the heart was made out. The second sound at the apex was accentuated, and there was a soft systolic murmur which was not transmitted. Abdominal examination showed the uterus enlarged and reaching to the level of the umbilicus—a finding inconsistent with her estimated duration of pregnancy. Fetal parts were not palpated, and no fetal heart tones could be heard. There was an old midline scar in the lower abdomen which was solidly healed. There was moderate tenderness on palpation over the right lower quadrant of the abdomen, but no spasm. A placental souffle was heard in the left lower quadrant. The red blood-cell count was 4,610,000 (Sahli), the white blood-cell count 16,350, and the hemoglobin 93 per cent.

Because of the history of massive hemorrhage twenty-eight hours previously, it was decided to exxamine the patient vaginally under sterile precautions as soon as compatible donors were available. These were obtained, and under nitrous-oxide-oxygen anesthesia a vaginal examination was performed. On introducing the finger through the cervix, which was slightly dilated, soft placenta-like tissue could be felt overlying the entire os. Believing this to be a complete placenta previa, it was decided to insert a Voorhees bag intraovularly. On attempting to pass the index finger through the placenta, it was found that this tissue filled the entire uterus. During this time the cervix had dilated with apparent ease to approximately 2.5 cm., and the patient was bleeding profusely. Examination of a portion of the tissue from the uterus revealed the characteristic grape-like vesicles of an hydatidiform mole. By finger curettage most of the mole was then evacuated from the uterus. An ampule of posterior pituitary extract was given intravenously without any apparent effect upon the bleeding.

By this time the patient was in profound surgical shock. The vagina was sponged out, and the cervix inspected. No lacerations were noted. The uterus was then packed, and the patient treated for shock. She was given an intravenous infusion of 800 cc. of 10 per cent glucose in saline solution, and this was closely followed with a transfusion of 800 cc. of citrated blood. Within thirty minutes a second transfusion of 600 cc. of citrated blood was administered. At this time the blood pressure was unobtainable, and the pulse rate obtained from the abdominal aorta ranged

A series of selected case histories by members of the section will be published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the section.

from 160 to 180 per minute. The uterus remained soft and boggy and was apparently filled up with blood. Therefore, 3 hours after removal of the mole a supravaginal hysterectomy was performed under cyclopropane anesthesia. The uterus was found to be large and soft and was obviously filled with recently clotted blood. The patient appeared to be no worse following the hysterectomy, which required about one-half hour. She was given 2 ampules, 50 cc., each of 7.5 per cent sodium bicarbonate solution intravenously and another transfusion of 500 cc. of citrated blood. The blood pressure was then obtainable for the first time since delivery and was systolic 60, diastolic 40. She continued to receive one 50 cc. ampule of sodium bicarbonate solution every twenty minutes for a total of six doses. One hour following the last transfusion 700 cc. of citrated blood was given, and the blood pressure slowly rose to systolic 90, diastolic 50.

It was discovered at this time that there was still a continuous trickle of blood from the vagina, and a vaginal examination was again performed under sterile precautions. The bleeding point could not be located even with the aid of a speculum, and the vagina was packed with sterile gauze. This procedure caused some reaction, and the patient again went into shock. She was placed on continuous intravenous administrations, including citrated blood, and saline and soda bicarbonate solutions, and in three hours the blood pressure increased to systolic 110, diastolic 70. During this time the fifth transfusion of 700 cc. of citrated blood was given. The pulse rate, palpable at the wrist, ranged from 120 to 140 per minute, and the systolic blood pressure ranged from 100 to 120. The vaginal packing had been stained through, but blood was not oozing. Seven hours after delivery when the patient had had 3,300 cc. of citrated blood, 800 cc. of 10 per cent glucose solution, 1,250 cc. of normal saline solution and 150 cc. of 7.5 per cent sodium bicarbonate solution—a total of 5,500 cc. of fluid by vein—the cannula was removed from the right arm, and the patient transferred to bed. For the next six hours she slept under sedation and took considerable fluid by mouth. One hour later the patient either coughed or retched, and expelled the vaginal pack. She began to bleed furiously from the vagina and lost between 500 and 700 cc. of blood. She again went into profound shock. Under nitrous-oxide anesthesia she was rapidly prepared and examined. Approximately 300 cc. of partially clotted blood was removed from the vagina. At this time a deep laceration of the cervix extending toward the right vault was found. This was repaired with interrupted catgut sutures, and the vagina was not repacked. During the operation the patient was transfused with 600 cc. of citrated blood and within one hour another 600 cc. transfusion was given. At the end of this transfusion the patient showed some evidence of pulmonary edema, and all intravenous fluids were stopped. Two hours after the hemorrhage her pulse rate was 140 per minute, and the blood pressure systolic 106, diastolic 52.

The patient made a surprisingly good recovery. She ran a febrile course for nine days, the temperature never rising above 101.5°F. After forty-eight hours, the pulse reached a level of 90 per minute, and the blood pressure rose to systolic 150, diastolic 90. On the twelfth postoperative day the temperature was 98.4°F., the pulse 100, and the respirations 20.

Comment. Massive hemorrhage from hydatidiform mole such as occurred in this case is unusual. More commonly there are several attacks of slight or moderate bleeding from the vagina, the amount of flow generally increasing until treatment is instituted. Some of the findings in this case were atypical, such as the history of fetal movements, the small size of the uterus for the period of gestation and the known lack of growth of the uterus over a period of three months.

In hydatidiform mole there is usually more rapid growth of the uterus than in normal pregnancy. Therefore it is not surprising that the diagnosis was not made before thorough vaginal exploration was performed. The rationale of having available blood donors present during operative procedures in bleeding cases was amply justified in this case. Without such precautions this patient would undoubtedly have been lost.

The chief omission in the handling of this case was the failure to make an earlier diagnosis of the cervical laceration which must have occurred during the finger curettage of the uterus. Inspection of the cervix with a speculum is inadequate in making a diagnosis of laceration of the cervix, because the lacerated edges may lie flat together so that the chief source of bleeding is missed. It is only by placing two or more forceps on the cervix so the lips are carefully spread under good light and with adequate retraction of the vaginal walls that a cervical laceration can be ruled out.

The use of the sodium bicarbonate solution was based on the work of Coonse et al.* on the experimental treatment of shock. This case demonstrates excellent, prompt treatment of massive, continuous hemorrhage and shock with a satisfactory result.

*Coonse, G. K.; Foisie, P. S.; Robertson, H. F., and Aufranc, O. E.: Traumatic and hemorrhagic shock, experimental and clinical study. New Eng. J. Med. 212:647 (April 11) 1935.

RECENT DEATHS

RICE—Frederick W. Rice, M.D., of 16 Elko Street, Brighton, died August 31 at Cape Porpoise, Maine. He was in his seventy-second year.

Dr. Rice was born in Jamaica Plain and, after attending Dedham schools, was graduated in 1894 from New York University College of Medicine. He studied at the Boston Dispensary and the Bellevue Hospital, New York. He had served as a surgeon for the New York Central Railroad and for ten years had been Boston school physician. In addition, he served on the staffs of several local hospitals and maintained a practice in Brighton for forty-three years.

Dr. Rice was a fellow of the Massachusetts Medical Society and the American Medical Association.

His widow, three daughters, a son, and a grandson survive him.

FOGERTY—William C. Fogerty, M.D., of 98 Chandler Street, Worcester, died August 31 at his home. Dr. Fogerty was in his seventy-ninth year.

A native of Worcester, he graduated from Dennison Medical College and subsequently served a four-year internship at Philadelphia General Hospital. Dr. Fogerty retired in 1935 after having served fifty-four years as a general practitioner in Worcester.

He was a fellow of the Massachusetts Medical Society and a member of the American Medical Association.

His widow, a daughter, and two brothers, survive him.

WARREN—Arthur Fay Warren, M.D., of Chicopee Falls, died suddenly August 16. He was in his sixty-third year.

A native of Monson, Dr. Warren graduated from Milford, N. H., High School, Worcester Academy and from the New York Homeopathic Medical School with honors.

Beginning his practice in Winchendon, he then moved to Belchertown, and from there went to Chicopee Falls where he remained. Dr. Warren served on the staffs of the Wesson Memorial and Maternity hospitals.

His widow, Mrs. Katherine Warren, a daughter, Mrs. Katherine W. Ramsey, two grandchildren, and one brother, Paul K., survive him.

OBITUARY

DR. GEORGE HENRY SIMMONS

Dr. George H. Simmons, editor and general manager emeritus of the *Journal of the American Medical Association,* died in St. Luke's Hospital, Chicago, September 1, following an operation performed August 25.

Dr. Simmons was born in Morton, England, January 2, 1852. He came to the United States in 1870 and studied at Tabor College in Iowa in 1871 and 1872, and at the University of Nebraska from 1872 to 1876. While a student in the Nebraska University, Dr. Simmons acted as editor of the *Nebraska Farmer,* assistant editor of the *Nebraska State Journal* and field correspondent of the *Omaha Republican,* and *Kansas City Journal.* His editorial ability was recognized in these several fields and even when a freshman in the university he won a prize for an essay on the sheep industry. From these varied activities, he secured means for meeting the expenses of his subsequent education.

He was awarded the M.D. degree from the Hahnemann Medical College of Chicago in 1882 and, following service at the Rotunda Hospital in Dublin in 1884, he studied at Rush Medical College and was given another M.D. degree in 1892.

From 1884 to 1899 he practiced medicine in Lincoln, Nebraska, and in 1896 established the *Western Medical Review,* acting as its editor. From 1895 to 1899 he served as secretary of the Nebraska State Medical Society and of the Western Surgical and Gynecological Society.

In political affairs he demonstrated qualities of leadership and brought reforms in the state government of Nebraska.

In 1899 Dr. Simmons was selected by the Board of Trustees of the American Medical Association to be the general secretary of this organization, filling the position until 1911 and in addition serving as editor of its *Journal* until 1924. These appointments were made after a comparative study of the qualifications of a large group of prominent applicants for these positions. During his years as incumbent, he inaugurated the movement to reorganize the administration of the medical and business affairs of the Association and in 1901 was made general manager in addition to his other duties. The phenomenal growth of the Association and its publications during the succeeding years of Dr. Simmons' administration are tributes to his energy and sound judgment.

The more notable changes which took place up to 1924 under the leadership of this dynamic personality are the initiation of movements for the advancement of medical education and science, the publication of information concerning medical schools, co-operation with state boards of medical licensure, the organization of the Council on Medical Education and Hospitals, association with the Council on Pharmacy and Chemistry, the organization of the Department of Propaganda for Reform which later became the Bureau of Investigation, the publication of the American Medical Directory and several journals devoted to special departments of practice, the *Quarterly Cumulative Index Medicus* and *Hygeia.*

Despite these complicated and varied activities Dr. Simmons was also devoted to the problems incident to army medical service. He was commissioned first lieutenant in the Medical Reserve Corps of the United States Army in 1908, and in 1917 was made a major in the Personnel Division. In 1921, by order of President Harding, he was awarded the Distinguished Service Medal.

His successor, Dr. Morris Fishbein, writes of Dr. Simmons as follows:

"As an editor, Dr. George H. Simmons was alert and fearless. His attacks on quackery and fraud in the field of medicine brought on his unwearying head and shoulders the counterattacks of those who saw their unscrupulous exploitations exposed and their incomes discontinued. It was his policy never to reply to any of the personal attacks made on him in the course of his service. Innumerable medical writers could testify to the manner in which he devoted himself personally to the education of younger men in editorial technic. . . .

"His work for the American Medical Association was characterized by intelligence, unselfishness, initiative and righteousness. In his personal life he had his share of physical and mental suffering. He weathered storms of unjust criticism and false characterization of his administration. He devoted himself almost objectively and in a manner completely devoid of personal interest to the public career which he had chosen. Unquestionably he was the greatest factor in his generation in the development of the American Medical Association and the profession that it represents."

A former editor of the *New England Journal of Medicine* enjoyed the privilege of meeting Dr. Simmons on several occasions and was impressed by his modesty and aversion to any reference to his accomplishments.

Life to him had been in the largest sense an opportunity for service to human welfare.

NOTICES

ANNOUNCEMENT

NIELS L. ANTHONISEN, M.D., announces the opening of an office at 82 Marlborough Street, Boston. Telephone Commonwealth 4142.

BOSTON SOCIETY OF ANESTHETISTS

The next meeting of the Boston Society of Anesthetists will be held at the Hotel Kenmore, Tuesday, September 14, at 8:30 p. m.

The program will consist of round-table reports of interesting cases and methods.

Physicians and medical students are cordially invited to attend.

PHILIP D. WOODBRIDGE, M.D., *President.*

PENTUCKET ASSOCIATION OF PHYSICIANS

There will be a meeting of the Pentucket Association of Physicians, September 16, at "Duck In," at 8:30 p. m., tel. Merrimac 280. Joel E. Goldthwait, M.D., will be the speaker. His subject will be "Problem of the Arthritic."

THE FOUR-COUNTY MEDICAL SOCIETY

The next meeting of the Four-County Medical Society will be held on Wednesday, September 15, at Northampton.

There will be medical and surgical clinics at the Cooley Dickinson Hospital at 9:00 a. m. A luncheon will be served at the Hotel Northampton at 12:00 noon.

The guest speaker will be Dr. Channing Frothingham of Boston, president of the Massachusetts Medical Society. He has chosen as his subject "How Shall We Meet Present Trends in Medicine?" Dr. Paul M. Ashton, of Springfield, will speak on "The Practical Use of Endocrines in Gynecology" and Dr. Stanley S. Cox of Holyoke on "Traumatic Surgery."

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, SEPTEMBER 13

TUESDAY, SEPTEMBER 14—

8:30 p. m. Boston Society of Anesthetists.

WEDNESDAY, SEPTEMBER 15—

†12 m. Clinicopathological conference. Children's Hospital Amphitheater.

FRIDAY, SEPTEMBER 17—

12 m. Massachusetts General Hospital. Clinical meeting of the Staff of the Children's Medical Service. Ether Dome.

SATURDAY, SEPTEMBER 11—

•10 a. m. - 12 m. Staff rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

•Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

SEPTEMBER 13-17—Fifth International Congress of Radiology. Page 709, issue of April 22, and page 233, issue of August 5.

SEPTEMBER 14—Boston Society of Anesthetists. Notice above.

SEPTEMBER 15—The Four-County Medical Society. Notice above.

SEPTEMBER 16—Pentucket Association of Physicians. Notice above.

SEPTEMBER 20-24—Sixteenth Annual Session of the American Congress of Physical Therapy. Page 288, issue of August 12.

SEPTEMBER 21, 22 and 23—Clinical Congress of the Connecticut State Medical Society, New Haven. Page 237, issue of August 5.

OCTOBER 1 and 2—New England Surgical Society. Providence, R. I.

OCTOBER 3-16—Medicomilitary Inactive Duty Training. Page 280, issue of August 12.

OCTOBER 5-8—American Public Health Association meeting. New York City.

OCTOBER 9—American Board of Ophthalmology, Chicago. Page 722, issue of April 22.

OCTOBER 14-16—Association of Military Surgeons of the United States. Page 379, issue of August 26.

OCTOBER 18-22—Interstate Postgraduate Medical Association. Page 418, issue of September 2.

OCTOBER 19, 20 and 21—Academy of Physical Medicine. Page 723, issue of April 22, and page 288, issue of August 12.

OCTOBER 25-29—American College of Surgeons. Chicago, Illinois.

NOVEMBER 1-12—1937 Graduate Fortnight of the New York Academy of Medicine. Page 1000, issue of June 3.

NOVEMBER 6—American Board of Obstetrics and Gynecology. Page 153, issue of July 22.

APRIL 4-8, 1938—The American College of Physicians. Page 41, issue of July 1.

JUNE 13 and 14, 1938—American Board of Obstetrics and Gynecology. Page 153, issue of July 22.

DISTRICT MEDICAL SOCIETIES

BRISTOL SOUTH DISTRICT MEDICAL SOCIETY

NOVEMBER 4—5 p. m., Fall River.

MAY 5, 1938—5 p. m., New Bedford.

FRANKLIN DISTRICT MEDICAL SOCIETY

SEPTEMBER 14—Franklin County Hospital, Greenfield, 11 a. m.

Meetings will be held at the Franklin County Hospital, Greenfield, at 11 a. m. the second Tuesdays of November, January, March and May.

WORCESTER DISTRICT MEDICAL SOCIETY

At the following meetings, except the annual meeting, dinner will be at 6:15, to be followed by business session and scientific program.

OCTOBER 13—Rutland State Sanatorium, Rutland.

NOVEMBER 10—Grafton State Hospital, North Grafton.

DECEMBER 8—St. Vincent Hospital, Worcester.

JANUARY 12—Worcester City Hospital, Worcester.

FEBRUARY 9—Worcester State Hospital, Worcester.

MARCH 9—Memorial Hospital, Worcester.

APRIL 13—Hahnemann Hospital, Worcester.

MAY 11—Afternoon and evening, annual meeting. Place and schedule of the program to be announced.

BOOK REVIEW

Transactions of the American Gynecological Society. Volume 61 for the year 1936. Edited by Otto H. Schwartz. 376 pp. St. Louis: C. V. Mosby Company, 1937.

This is an exceedingly well and handsomely bound volume containing twenty-three papers read at the annual meeting of the American Gynecological Society in 1936. Because of the excellence of each contribution, the fact that they have appeared individually in periodicals does not detract from the value of the collection. These are an example of what professors of gynecology and obstetrics in our own medical schools and of what heads of the clinical departments of gynecology and obstetrics in some of our best hospitals are doing to enlighten their field. The beam from their studious researches is thrown too widely over the field to permit here even a table of contents. Suffice it to say that this collection has real value for all students of gynecology and obstetrics.